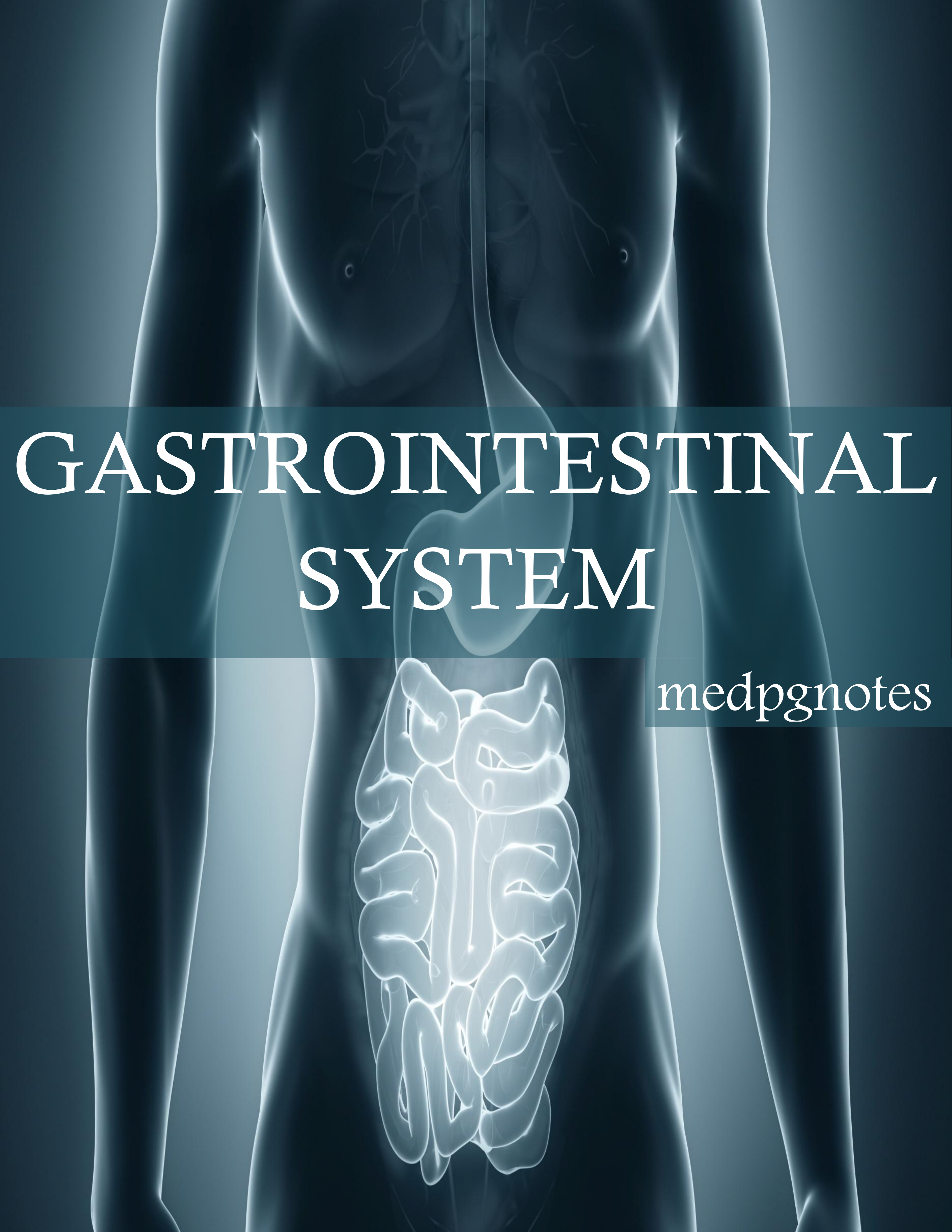

GASTROINTESTINAL SYSTEM

medpgnotes

GASTROINTESTINAL SYSTEM

CONTENTS

DEVELOPMENT OF GASTROINTESTINAL SYSTEM 9

ANATOMY OF GASTROINTESTINAL SYSTEM 9

GENERAL FEATURES OF ANATOMY OF GASTROINTESTINAL SYSTEM 9

ANATOMY OF OMENTUM AND MESENTRY 9

PHYSIOLOGY OF GASTROINTESTINAL SYSTEM 10

GASTROINTESTINAL SECRETIONS 10

GASTROINTESTINAL ENZYMES AND HORMONES 11

REGULATION OF GASTROINTESTINAL SYSTEM 13

ABSORPTION IN GASTROINTESTINAL SYSTEM 14

GENERAL FEATURES OF GASTROINTESTINAL SYSTEM 14

ESOPHAGUS 15

ANATOMY OF ESOPHAGUS 15

GENERAL FEATURES OF ESOPHAGUS 16

ESOPHAGEAL ATRESIA 16

TRACHEOESOPHAGEAL FISTULA 16

DYSPHAGIA 17

ACHALASIA CARDIA 17

DIFFUSE ESOPHAGEAL SPASM 18

ESOPHAGEAL RING 18

ESOPHAGITIS 18

BARRET'S ESOPHAGUS 19

CARCINOMA ESOPHAGUS 19

MALLORY WEISS SYNDROME 20

HIATUS HERNIA 20

GASTROESOPHAGEAL REFLUX DISORDER 21

ESOPHAGEAL PERFORATION 21

BOERRHAVE SYNDROME 22

UPPER GI BLEED 22

STOMACH AND SMALL INTESTINE 23

ANATOMY OF STOMACH 23

ANATOMY OF SMALL INTESTINE 23

GENERAL FEATURES OF STOMACH AND SMALL INTESTINE 24

ACUTE DILATATION OF STOMACH 25

PEPTIC ULCER 25

H.PYLORI 26

GASTRIC ULCER 27

DUODENAL ULCER 29

DUODENAL STRICTURE 29

DUMPING SYNDROME AND VAGOTOMY 30

POST CIBAL SYNDROME 30

GASTRIC OUTLET OBSTRUCTION 30

MENETRIER DISEASE 31

FEATURES OF CARCINOMA STOMACH 31

MANAGEMENT OF CARCINOMA STOMACH 32

HYPERTROPHIC PYLORIC STENOSIS 33

POLYPOSIS 34

GASTRIC DIVERTICULA 35

GASTRIC FISTULA 35

GASTROINTESTINAL STROMAL MALIGNANCY 35

SMALL INTESTINE TUMOURS 35

PEUTZ JEGHER POLYPOSIS 36

LARGE INTESTINE 36

GENERAL FEATURES OF LARGE INTESTINE 36

LOWER GI BLEEDING 38

FEATURES OF CARCINOMA COLON 38

DIAGNOSIS OF CARCINOMA COLON 39

TREATMENT OF CARCINOMA COLON 39

FEATURES OF CARCINOMA RECTUM 40

MANAGEMENT OF CARCINOMA RECTUM 40

CARCINOMA ANAL CANAL 40

HIRSCHPRUNG'S DISEASE 41

DIVERTICULOSIS AND DIVERTICULITIS 42

ANGIODYSPLASIA 42

MECKEL'S DIVERTICULUM 43

INTESTINAL TUBERCULOSIS 43

RECTAL ULCER 44

RECTAL POLYP ... 44
RECTAL PROLAPSE ... 44
ANATOMY OF ANAL CANAL ... 45
PILONIDAL SINUS ... 45
HEMORRHOIDS ... 46
ANAL FISSURE ... 46
FISTULA IN ANO ... 46
ANORECTAL ANOMALIES ... 47
ANORECTAL AND PERIANAL ABSCESS ... 47
SUPERIOR MESENTERIC ARTERY SYNDROME ... 47
PNEUMATOSIS INTESTINALIS ... 48
INTESTINAL STRICTURES ... 48
INTESTINAL MANIFESTATIONS OF TYPHOID ... 48
COLONOSCOPY ... 49
ILEOSTOMY AND COLOSTOMY ... 49
INTESTINAL AMOEBIASIS ... 49

DIARRHOEA AND CONSTIPATION ... 49
  IRRITABLE BOWEL SYNDROME ... 49
  DIARRHEA ... 49
  DEHYDRATION ... 51
  ORAL REHYDRATION SALT ... 52
  CONSTIPATION ... 52

APPENDIX ... 52
  GENERAL FEATURES OF APPENDIX ... 52
  APPENDICITIS ... 53
  TUMOURS OF APPENDIX ... 54

INFLAMMATORY BOWEL DISEASE ... 54
  GENERAL FEATURES OF INFLAMMATORY BOWEL DISEASE ... 54
  CROHN’S DISEASE ... 54
  ULCERATIVE COLITIS ... 55

INTESTINAL OBSTRUCTION ... 57
  GENERAL FEATURES OF INTESTINAL OBSTRUCTION ... 57
  INTESTINAL ATRESIA ... 58
  MALROTATION ... 58

PARALYTIC ILEUS 59

MECONIUM ILEUS 59

INTUSUSSCEPTION 59

VOLVOLUS 60

INTESTINAL PSEUDO OBSTRUCTION 61

DIAGNOSIS OF INTESTINAL OBSTRUCTION 61

MALABSORPTION SYNDROMES 61

GENERAL FEATURES OF MALABSORPTION 61

D-XYLOSE TEST 62

CELIAC DISEASE 62

WHIPPLE DISEASE 63

SHORT BOWEL SYNDROME 63

TROPICAL SPRUE 64

PROTEIN LOSING ENTEROPATHY 64

LIVER 64

ANATOMY AND PHYSIOLOGY OF LIVER 64

LOBES OF LIVER 65

GENERAL FEATURES OF LIVER 66

LIVER ENZYMES 67

UNCONJUGATED HYPERBILIRUBINEMIA 67

CONJUGATED HYPERBILIRUBINEMIA 68

JAUNDICE 69

CHOLESTASIS 69

CRIGGLER NAJJAR SYNDROME 70

GILBERT SYNDROME 70

DUBIN JOHNSON SYNDROME 70

ROTOR SYNDROME 70

HEMOBILIA 70

REYE SYNDROME 71

FATTY LIVER 71

MIRIZZI SYNDROME 71

GENERAL FEATURES OF HEPATITIS 71

HEPATITIS A 72

FEATURES OF HEPATITIS B 72

GASTROINTESTINAL SYSTEM

DIAGNOSIS OF HEPATITIS B 73
TREATMENT OF HEPATITIS B 74
FEATURES OF HEPATITIS C 75
MANAGEMENT OF HEPATITIS C 75
HEPATITIS D 76
HEPATITIS E 76
HEPATITIS G VIRUS 76
CHRONIC HEPATITIS 76
ALCOHOLIC HEPATITIS 77
AUTOIMMUNE HEPATITIS 77
NON ALCOHOLIC FATTY LIVER DISEASE 77
ASCITES 78
FEATURES OF HEPATIC FAILURE 78
MANAGEMENT OF HEPATIC FAILURE 79
HEPATORENAL SYNDROME 79
FEATURES OF HEPATOCELLULAR CARCINOMA 79
FIBROLAMELLAR VARIANT OF HEPATOCELLULAR CARCINOMA 80
DIAGNOSIS OF HEPATOCELLULAR CARCINOMA 80
TREATMENT OF HEPATOCELLULAR CARCINOMA 80
WILSON’S DISEASE 80
HEMOCHROMATOSIS 81
HYDATID CYST OF LIVER 82
PYOGENIC LIVER ABSCESS 82
AMOEBIC LIVER ABSCESS 83
HEPATIC ENCEPHALOPATHY 83
LIVER TRANSPLANTATION 83
CIRRHOSIS 84
BILIARY CIRRHOSIS 85
NON CIRRHOTIC PORTAL FIBROSIS 85
PORTAL HYPERTENSION 85
ESOPHAGEAL VARICES 86
NODULAR REGENERATIVE HYPERPLASIA 87
FOCAL NODULAR HYPERPLASIA 87
EXTRAHEPATIC PORTAL VEIN OBSTRUCTION 87

GASTROINTESTINAL SYSTEM

GALL BLADDER 88
  GENERAL FEATURES OF GALL BLADDER 88
  FEATURES OF GALL STONES 88
  DIAGNOSIS OF GALL STONES 89
  TREATMENT OF GALL STONES 89
  FEATURES OF CHOLECYSTITIS 90
  DIAGNOSIS OF CHOLECYSTITIS 90
  TREATMENT OF CHOLECYSTITIS 90
  CARCINOMA GALL BLADDER 91
BILIARY TRACT 91
  ANATOMY OF BILIARY TRACT 91
  FEATURES OF BILIARY TRACT 92
  CHOLANGITIS 92
  FEATURES OF COMMON BILE DUCT STONES 93
  DIAGNOSIS OF COMMON BILE DUCT STONES 93
  TREATMENT OF COMMON BILE DUCT STONES 94
  BILE DUCT INJURIES 94
  BILIARY ATRESIA 95
  CHOLEDOCHAL CYST 95
  BILIARY STRICTURE 95
  BILIARY FISTULA 96
  CHOLANGIOCARCINOMA 96
  PNEUMOBILIA 96
SPLEEN 97
  ANATOMY OF SPLEEN 97
  FEATURES OF SPLEEN 97
  SPLENOMEGALY 98
  SPLENECTOMY 98
  SPLENIC INJURIES 98
  SPLENIC RUPTURE 99
PANCREAS 99
  ANATOMY OF PANCREAS 99
  FEATURES OF PANCREAS 99
  FEATURES OF ACUTE PANCREATITIS 100

DIAGNOSIS OF ACUTE PANCREATITIS 101

TREATMENT OF ACUTE PANCREATITIS 102

FEATURES OF CHRONIC PANCREATITIS 102

MANAGEMENT OF CHRONIC PANCREATITIS 102

PSEUDOCYST OF PANCREAS 102

HEMORRHAGIC PANCREATITIS 103

FEATURES OF CARCINOMA PANCREAS 103

MANAGEMENT OF CARCINOMA PANCREAS 104

PERIAMPULLARY CARCINOMA 104

PANCREATIC INJURIES 104

FEATURES OF GASTRINOMA 105

MANAGEMENT OF GASTRINOMA 105

FEATURES OF INSULINOMA 106

MANAGEMENT OF INSULINOMA 106

GLUCAGONOMA 107

MUCOVISCOIDOSIS 107

ANNULAR PANCREAS 107

PANCREATIC CHOLERA 107

DRUGS ACTING ON GASTROINTESTINAL TRACT 107

KEY TO THIS DOCUMENT

Text in normal font – Must read point. Asked in any previous medical entrance examinations

Text in bold font – Point from Harrison's text book of internal medicine 18th edition

Text in italic font – Can be read if you are thorough with above two

DEVELOPMENT OF GASTROINTESTINAL SYSTEM

Features of development of Gastrointestinal system	Lower respiratory system develops from foregut Stomach rotates 90* clockwise and its posterior wall grows faster, Duodenum rotates to right and its retroperitoneal, Dorsal bud forms accessory pancreatic duct of santorinin, Dorsal duct forms santorini duct, Ventral bud forms uncinate process and inferior part of head of pancreas
Derivatives of Ventral Mesogastrium	Coronary Ligament, Falciparum Ligament, Lesser Omentum
NOT a Derivative of Ventral Mesogastrium	Gastrosplenic Ligament
Spleen develops from	*Dorsal mesogastrium*
Uncinate process is formed from	*Ventral pancreatic bud*
Derivatives of septum transversum	Falciform ligament, Coronary ligament, Mesentery of lesser sac
Kupffer cells are formed from	*Septum transversum*
Organ NOT developing in mesentry of stomach	Kidney
Umbilical vein assume which position after birth	Ligamentum teres
Vessel as a remnant structure	Ligamentum teres
Teres major of liver is a remnant of	Left umbilical vein
Ductus venosus connect	Umbilical vein to sinus venosus
Third part of Duodenum develops from	Midgut

ANATOMY OF GASTROINTESTINAL SYSTEM

GENERAL FEATURES OF ANATOMY OF GASTROINTESTINAL SYSTEM

Nerve plexus located between Longitudinal and circular fibres of GIT	Myenteric/Auerbach's Plexus
Meissner's plexus are located in	Submucosal region
Temporary mucosal fold	Longitudinal rectal fold
Submucosal glands are found in	Duodenum
GALT is primarily located in	Lamina propria
Centre of villi has a terminal lymphatic duct called	*Lacteal*
Ligament of treitz	*Supports duodenojejunal flexure*

ANATOMY OF OMENTUM AND MESENTRY

Dorsal mesentry of stomach	*Greater omentum*
Ventral mesentry of stomach	*Lesser omentum*
Mesentry of small intestine along its attachment to posterior abdominal wall crosses	Third part of duodenum, aorta, right urerter

Root of mesentry crosses	Right ureter, right psoas major, right testicular artery, abdominal aorta, inferior vena cava
Root of mesentry crossed by	Horizontal part of duodenum, Third part of duodenum, Aorta, Right ureter
Root of mesentry does NOT cross	Left gonadal vessels
Boundaries of lesser sac	Greater omentum, Posterior wall of stomach
Contents of lesser omentum	Hepatic artery, Portal vein, Bile duct
Not a content of Lesser omentum	Stomach, Inferior Vena cava
NOT a content of Lesser Omentum	Hepatic Vein, IVC
Structures attached to right free border of lesser omentum	*Hepatic artery, portal vein, common bile duct*
Structures involved while resecting free edge of lesser omentum	Portal vein, Hepatic artery , Common bile duct
Location of Omental bursa	Left Subhepatic
Intestine commonly strangulated in	Omental bursa
Relation to epiploic foramen	Bile duct anteriorly
Foramen of winslow	Between greater sac and lesser sac
Boundaries of epiploic foramen	*Caudate process of liver, inferior vena cava, free edge of lesser omentum, first part of duodenum*
NOT a boundary of epiploic foramen	*Quadrate lobe*
Milky spots on Omentum are rich in	Macrophages
Folds on distal hepatopancreatic duct	*Plicae circularis*
Superior retention band in adults	*Ligament of trietz*
Inferior retention band in adults	*Phrenicocolic ligament*
Ligament of trietz	*Suspensory muscle of duodenum, holds duodenum open or closed*
NOT true about ligament of trietz	It is a part of mesentery
Content of epiplocele	Omentum
Left colic artery lies on	*Descending mesocolon*

PHYSIOLOGY OF GASTROINTESTINAL SYSTEM

GASTROINTESTINAL SECRETIONS

Volume of saliva per day	*1500 ml*
Predominant ion in saliva under resting conditions	Bicarbonate
Volume of gastric secretion per day	*2000 ml*
Acid secretion is stomach	Gastrin increases it, Secretin decreases it, H2 blocker decrease it, Total acid secretion reflects functional parietal cell mass, Total acid output indicates parietal cell mass activity
Acid secretion in stomach is NOT increased by	Prostaglandins
Gastric secretion stimulated by	Gastrin, Histamine, Vagal stimulus, Gastric distension
Parietal cells secrete	HCL
Action of HCL	Activation of Pepsinogen to Pepsin
Produced by stomach and has a role in control of food	Ghrelin

intake	
GIP is secreted by	*K cells*
Normal gastric juice does NOT have	Ca++
Mucin is NOT secreted by	Paneth cells
Volume of pancreatic secretion per day	*1500 ml*
Very high pH	Pancreatic juice
Maximum pH is seen in	Pancreatic secretions
Alkalinity of pancreatic juice is associated with	*Intercalated ducts*
Pancreatic secretion contains	Trypsin , Lipase
Pancreatic juice rich in water and electrolytes poor in enzymes is secreted in response to	Secretin
Pancreatic juice	Isoosmotic with blood plasma, Secretes 1 ½ litre of alkaline fluid daily, Secretion is rich in enzymes
Pancreatic juice rich in water and electrolytes but poor in enzymes is secreted in response to	Secretin
NOT present in pancreatic juice	*Aminopeptidase*
Main substrate for Pancreatic lipase	Triacylglycerol
End products of pancreatic lipase digestion	Fatty acids
Enterohepatic circulation is necessary for secretion of	Bile
Volume of biliary secretion per day	*500 ml*
Bile acid has detergent action due to	Amphipathic nature of bile acid
Bile acid uptake by hepatocyte is dependent upon	*Sodium*
Most important stimulus for bile secretion	Bile salt
Bilirubin is secreted by	Bile salts
Ingested triglycerides are emulsified in intestine by	Bile salts
Fat is kept in solubility by	Bile salts
Bile salts are produced from	Cholesterol
Emulsifiers	Bile salts
After a carbohydrate rich meal, insulin secretion is stimulated by	GLP 1 (7-36 amide)

GASTROINTESTINAL ENZYMES AND HORMONES

NOT secreted in proenzyme form	Ribonuclease
Hormone NOT secreted by alimentary tract	Chymotrypsin
Pepsinogen is activated by	Low pH
Stable at acidic pH	Pepsin
Pepsin is activated by	HCL
Gastrin is a	Polypeptide
Gastrin is produced by	Pancreas, gastric antral cells and pituitary
Gastrin is produced in	Pyloric antrum
Secretin family	*Secretin, VIP, glucagon*
Secretin	Inhibits gastric emptying, Increases bicarbonate rich pancreatic secretion, Potentiates action of CCK, Increases bile secretion, Does NOT increase bile salts and bile acids, Contraction of pyloric sphincter
Secretin on Pancreatic juice	Increase Bicarbonate

Secretin is secreted by	Enterochromaffin like cells
Main stimulus for Secretin	Protein Degradation Products
Potential stimulus for release of secretin	Acidity of chyme
Secretin do NOT cause	Increase in gastric secretion
Best stimulus for secretin	Acid
Parietal cells/ Oxyntic cells are present in	**Neck/Isthumus**
Intrinsic factor secreted by	Oxyntic cells, Parietal cells
Neurotensin is secreted by	Ileum & Jejunum
Succus entericus is secreted by	Small intestine
Properties of hormone secreted by duodenum	Causes copious pancreatic juice rich in bicarbonate and poor in enzymes, Causes gall bladder to contract and sphincter of Oddi to relax, Leads to meager flow of pancreatic juice rich in enzymes
NOT a function of hormone secreted by duodenum	Increases gastric motility
Carboxypeptidase	*Exopeptidase*
Starch is converted to maltose by	*Amylase, pancreatic lipase, esterase*
Sucrose does not digest	*Isomaltose*
Most important enzyme for digestion of triglyceride	*Pancreatic lipase*
Chymotrypsinogen is activated into chymotrypsin by	Trypsin
Chymopepsinogen activation needs	Proteolytic enzyme
Chymotrypsinogen is a	Zymogen
Deficiency of enteropeptidase (enterokinase) affect	Trypsin
Enzyme NOT active within cell	Trypsin
Trypsin acts in	Alkaline medium
Function of trypsin and chymotrypsin	*Converts larger polypeptide to smaller ones*
Pancreatic enzyme which does NOT require trypsin as cofactor	*Ribonuclease*
Trypsinogen is converted to trypsin by	Enterokinase
Trypsin activator	Enterokinase
Autodigestion of pancreas is prevented by	*Trypsin inhibitors*
Trypsin inhibitors	Alpha 1 antitrypsin, Alpha 1 antiproteinase, Egg white
NOT a trypsin inhibitor	Enterokinase
Alpha 1 antitrypsin inhibits	**Serine protease, neutrophil elastase**
Main function of Cholecystokinin	Increases amylase secretion from pancreas
Best stimulus for CCK	Protein
CCK-PZ cause	Gall bladder contraction, Pancreatic enzyme secretion, Decreased lower esophageal sphincter
Actions of cholecystokinin	Contraction of gall bladder, Secretion of pancreatic juice rich in enzymes, Stimulates gastric emptying
NOT an action of cholecystokinin	Increases secretion of enterokinase, Augments action of secretion of gastrin
CCK related peptide from	**Duodenum**
Amylin secreted by	Beta cells
Alpha amylase	Cleaves only at alpha (1,4)
Inhibitory neurotransmitters in enteric neurons (gastrointestinal smooth muscle relaxants)	VIP, PACAP, NO
Required for digestion of dietary fat	Colipase, Bile salts, Pancreatic lipase
NOT a product of lipid digestion	*Diacylglycerol*

Primary function of gut flora	Synthesis of short chain fatty acids, Production of vitamin K, Fermentation of mucin
NOT an endopeptidase	Aminopeptidase

REGULATION OF GASTROINTESTINAL SYSTEM

Systems involved in gastrointestinal regulation	*Endocrine secretion, paracrine secretion, autonomous enteric nervous system*
Center for swallowing	*Nucleus tractus cuneatus*
Cephalic phase of gastric secretion is brought about by	Parasympathetic
Gastric phase of gastric secretion is brought about by	Hormonal factors
Gastric motility increased in	Acid in duodenum
Gastric motility decreased by	*Dopamine*
Basal electrical rhythm of stomach	*4/min*
Basal electrical rhythm of duodenum	*12/min*
Basal electrical rhythm of sigmoid colon	*16/min*
Migrating motor complex in humans	*Requires an intact intrinsic nervous system for coordinated propagation*
Migrating motor complex repeat at an interval of	*90/min*
First part of a test meal reaches splenic flexure by	*9 hours*
Two basic waves in smooth muscles of GIT	*Slow waves and spike waves*
Mixing waves of stomach is formed from	*Body of stomach*
Intestinal motility	Depend on gastric motility, Increased by distension, acetylcholine and cholecystokinin
Intestinal motility depends on	Gastric motility
Rate of gastric emptying increases with increase in	*Intragastric volume*
Gastric emptying decreased by	Cholecystokinin
Stimulation for gastric emptying	Gastrin
Gastrocolic reflex is related to	Mass peristalsis
Enterogastric reflex is stimulated by	Hyperosmolality of small intestine, Distension of duodenum, Products of protein digestion in duodenum, H+ ions bathing duodenal mucosa
NOT a stimulation for enterogastric reflex	Alkaline content of small intestine, Hormones
Pacemaker of GIT is located in	Pyloric end of stomach
Major regulator of interdigestive myoelectric complexes	Motilin
Small intestinal peristalsis is controlled by	Myenteric plexus
Chyme is propelled forward in small intestine by	Peristalsis
Maximum postprandial motility is seen in	Sigmoid colon
While doing sigmoidoscopy, if rectum is inflated with gas. Increased peristalsis is seen in	Distal colon
Maximum contraction of gall bladder is seen with	Cholecystokinin
Fat in duodenal lumen	Stimulates gall bladder contraction
Cholagagues cause	Contraction of Gall bladder
Increased blood flow during food digestion primarily increases because of	*Tissue hypertonicity and release of nitric oxide on to arterioles*

Vagal stimulation following intake of food does NOT affect secretion of	Gall bladder

ABSORPTION IN GASTROINTESTINAL SYSTEM

Events of absorptive state of the body is controlled by	*Insulin*
Amount of water absorbed in intestine per day	8 litres
Fastest to be absorbed from stomach	Water
Absorbed in proximal intestine	Iron
Iron absorption increased by	Vitamin C
Iron is actively absorbed from	Duodenum and proximal jejunum
Maximum absorption of water	Jejunum
Maximum absorption of folic acid	Jejunum
Maximum absorption of bile	Ileum
Vitamin B12 absorbed in	Ileum
Absorbed in colon	Electrolytes
Long and medium chain fatty acids are absorbed in	**Small intestine**
Short chain fatty acids produced by bacteria are maximally absorbed in	Colon
Longest transit time in GIT	Colon
Final sugars in intestinal chyme	Glucose and fructose
Only sugar absorbed in intestine against concentration gradient	Glucose
Absorption rate of sugars by small intestine is highest for	Hexose
Fructose is abosorbed by	**GLUT 5**
Fat absorption	Major fat absorption in small intestine, Fat in stool > 6 gm/day indicate malabsorption

GENERAL FEATURES OF GASTROINTESTINAL SYSTEM

Can present as acute abdomen	Tabes, Acute intermittent porphyria, Pneumonitis of lower lobe
Baseline investigation of acute abdomen in this high-tech era	X ray abdomen
Drugs causing radio opacity in bowel	Salicylate, Iron, Phenothiazine
Gasless abdomen	High obstruction, Acute pancreatitis, Congenital diaphragmatic hernia
DD for right upper lobe calcification	Gall stone, Renal stone, Calcification in vessels, Hepatic hemangioma
Colic generally disappears by	4 months
Commonest cause of abdominal pain in children	Warm colic
Recurrent abdominal pain is most often due to	Emotional problems
Vomiting on first day in a newborn is due to	Amniotic fluid gastritis
MC commensal gut flora in adult	Lactobacillus, Bacteriodes
In order to expose celiac axis, left renal artery, superior mesenteric artery and abdominal aorta in case of	Left medial visceral rotation

trauma	
Mattox Maneuver	Left Medial Visceral Rotation. Exposes Aorta, Celiac Axis, Superior Mesentric Artery, Left Renal Artery, Iliac Artery
Disparity of bowel ends anastomosis is corrected by	Cheatle maneuver
Cheatle split is done in	*Intestinal surgery*
Vascular anastamosis	Submucosa
VIPoma (Verner Morrison syndrome/Pancreatic cholera)	**Watery diarrhea, hypokalemia, achlorhydria (WDHA)**
VIPoma is common in	*Tail*
VIPoma	Hypokalemia, hypercalcemia, diarrhea
VIPoma	Diarrhoea, Muscle weakness history
NOT a feature of Verner Morrison syndrome	*Hyponatremia*
Do NOT cause GIT cancer	EBV
MC malignancy of GIT in children	Lymphoma
Offer protection from gastric aspiration syndrome in a patient with symptoms of flu	Metoclopramide
Gangrene of intestine is seen in	Shock, mesenteric artery thrombosis, volvolus
Immediate enema used in	Habitual constipation
Mesenteric vein thrombosis	Heparin is given, Surgery can lead to short bowel syndrome
Intestinal angina	Post prandial abdominal pain, weight loss, chronic mesenteric vessel ischemia
Bowel does NOT get strangulated in	Rectouterine pouch

ESOPHAGUS

ANATOMY OF ESOPHAGUS

Esophagus begins at	*C6*
Toughest layer of esophagus	Muscularis
Submucous glands are found in	Esophagus
Esophagus starts at	Lower end of cricoids
Esophagus	Length is 25 cm, Has squamous epithelium, Physiological sphincter located at lower end, Muscle layer in upper part is skeletal
Laimer Haeckerman triangle	*Between longitudinal and circular fibres of esophagus*
Press over esophagus	Aortic arch, Left atrium, Left bronchus
Does NOT press over esophagus	Right bronchus
Figures important for esophagist	15,25,40
Cervical constriction of esophagus at	Cricopharyngeus
Constrictions of esophagus at	25, 40
Appropriate constriction of diaphragm from nostril and incisor	17" and 16"
Second constriction in esophagus seen at	Crossing of aorta
Structure crossing esophagus at 25 cm from incisor teeth	Arch of aorta, Left bronchus

3rd constriction of esophagus is at the level of	Where it pierces diaphragm
Distance of gastroesophageal junction from upper incisor	40 cm
Gastroesophageal angle	*His angle*
Esophagus receive blood supply from	Bronchial artery, Inferior phrenic artery, Inferior thyroid artery
Esophagus does NOT receive blood supply from	Internal mammary artery
NOT a blood supply of esophagus	*Superior thyroid artery*

GENERAL FEATURES OF ESOPHAGUS

Incidence of congenital malformation in esophagus	1 in 4000
Secondary esophageal peristalsis	*Accompanied by lower esophageal sphincter relaxation*
Non progressive contraction of esophagus	Tertiary
Lower esophageal sphincter	Relaxes ahead of peristaltic wave
LES is relaxed by	Nitrates, Atropine, Calcium channel blockers, Alcohol, Caffeine/tea, Diazepam
Pressure of esophagus	7 mm below ambient pressure
Motility in upper third of esophagus decreased in	Pseudobulbar palsy, Chaga's disease, Myasthenia gravis
Contrast used for barium swallow	Barium Sulphate
Minimum stricture dilatation for swallowing	*16 mm*
Diffuse esophageal dilatation on barium swallow is seen in	Achalasia, Trypanosomiasis
Commonest benign tumor of esophagus	Leiomyoma
Foreign body usually arrested in esophagus	Above cricopharynx
Rigid esophagoscopy NOT done in	Cervical spine rigidity, aortic aneurysm
Treatment of nutcracker esophagus	*Long esophageal myotomy*

ESOPHAGEAL ATRESIA

One year old child with dyspnea and frothing from mouth with cyanosis	Esophageal atresia
Esophageal atresia is diagnosed by	Passing an orogastric tube
Absent stomach bubble on antenatal USG	Esophageal atresia of fetus
Contrast material used in diagnosis of esophageal atresia	Dianosil
Most common complication after primary esophagostomy for esophageal atresia with a distal tracheoesophageal fistula	*Gastroesophageal reflux*

TRACHEOESOPHAGEAL FISTULA

Newborn has dribbling after feeds. Respiratory distress and froth at mouth	Tracheoesophageal fistula

TEF showing gasless abdomen	EA with proximal TEF
H shaped tracheoesophageal fistula can be identified by	Esophagogram
Malignant tracheoesophageal fistula is best treated with	Surgical correction
Major determinant of poor survival in infants after tracheoesophageal fistula repair	*Presence of other anomalies*

DYSPHAGIA

Dysphagia may be due to	**Parkinson's disease**
Dysphagia Lusoria is due to	Aberrant Right Subclavian artery
Intermittent dysphagia	Pharyngeal pouch, Diffuse esophageal spasm
Odynophagia occurs in	Herpes esophagitis, Monilial esophagitis
A 60 year patient presenting with dysphagia of 6 weeks duration with solid foods. Now can swallow only liquids. diagnosis by	CXR, barium swallow, endoscopy, USG, CT scan
Initial Investigation esophageal dysphagia	Barium swallow
Young patient, dysphagia more to liquids than solids	Barium swallow
Investigation of choice for dysphagia for solids	Upper GI scopy
30 year female non progressive dysphagia both for solid and liquid	Multiple sacculation and pseudodiverticulae on barium swallow, corkscrew esophagus

ACHALASIA CARDIA

MC motility disorder causing dysphagia	Achalasia cardia
A 40 year old female presented with dysphagia to both liquids and solids and regurgitation for 3 months. Dysphagia was non progressive	Achalasia cardia
A 50 year old male Raju present with occasional dysphagia for solids, regurgitation of food and foul smelling breath. probable diagnosis	Achalasia cardia
Dysphagia for both solids and liquids. Bird's beak appearance in x ray	Achalasia cardia
Achalasia cardia	Dysphagia is the presenting symptom, Cause is absence of auerbach plexus, Pressure at distal end increased with no peristalsis
Maximum dilatation of Esophagus	Achalasia Cardia
Features of achalasia cardia	Increasing difficulty in swallowing more for liquids than for solids, regurgitant vomiting, Dilated and tortuous esophagus
Hurst phenomenon	Achalasia cardia
MC Complication of Achalasia cardia	Recurrent pulmonary infection
NOT true of Achalasia cardia	Mecholyl test is hyposensitive
NOT true about Achalasia cardia	Cork screw esophagus
NOT true about achalasia cardia	Mostly in women, Not premalignant
Amylnitrate inhalation test is useful in	Achalasia cardia
Pencil tip deformity	Achalasia cardia

Radiological feature of achalasia cardia	Absence of gastric air bubble, Air fluid level in mediastinum, Sigmoid esophagus
X ray finding suggestive of achalasia cardia	Megaesophagus, Tortuous esophagus, Fluid level in esophagus
Barium swallow showing dilatation with narrow smooth ending	Achalasia cardia
First investigation in Achalasia Cardia	Barium Swallow
NOT a radiological evidence of Achalasia cardia	Where it pierces diaphragm
Dysphagia for both solids and liquids. Barium swallow shows parrot beak appearance. increased LES pressure on esophageal manometry	Nitrates, Calcium channel blockers, Botulinum toxin, Myotomy
Treatment of achalasia cardia with high rate of recurrence	Botulinium toxin
Heller's operation done for	Achalasia cardia
NOT a treatment modality of Achalasia cardia	Colonic Interposition and anastomosis
NOT a treatment modality of achalasia cardia	*Calcium channel agonist*

DIFFUSE ESOPHAGEAL SPASM

Cork screw esophagus	Diffuse Esophageal Spasm
Investigation of choice in diffuse esophageal spasm	Manometry

ESOPHAGEAL RING

Non progressive dysphagia only with solids, barium study show proximal esophageal dilatation with distal constriction	Lower esophageal ring
Lower esophageal ring	**B ring**
MC cause of intermittent food impaction (Streakhouse syndrome)	**Schatazki ring**
Fibrous ring in lower esophagus	Schatazki ring
Schatazki ring	Mucosal ring at squamocolumnar junction, Dysphagia is presenting symptom, Causes dysphagia, Located at lower esophagus
Transfer dysphagia is seen in	**Schatazki ring**

ESOPHAGITIS

MC cause of esophagitis	Reflux disease
Barret's esophagitis leads to	Adenocarcinoma
Viruses that can cause esophagitis	HSV 1 and 2, HIV, Varicella zoster, HIV, CMV
Infective esophagitis is caused by	**Herpes, candida, CMV**
Treatment of candida esophagus	**Echinocandin**
Fenile esophagus	**Eosinophilic esophagitis, multiple circular ring, corrugated**
Best site of taking biopsy for viral esophagitis	Edge of ulcer (if CMV – base)

BARRET’S ESOPHAGUS

Etiology of Barrett’s ulcer	Ectopic gastric mucosa
Barrett’s esophagus show	Intestinal metaplasia
Barret esophagus is	Columnar metaplasia of lower third
Lining epithelium of Barrett’s esophagus	Columnar
A lesion 3 cm away from gastroesophageal junction containing columnar epithelium	Metaplasia
Columnar epithelium with goblet cells in lower esophagus in chronic heart burn patient	Metaplasia
Barret’s esophagus	Lower esophagus lined by columnar epithelium, Consequence of prolonged GER, Premalignant, Diagnosis by endoscopy and biopsy, Present as patchy or ring involvement
Adenocarcinoma of esophagus commonly found in	Barrett’s esophagus
Barrett’s Esophagus does NOT develop into	Squamous Cell Carcinoma
Barrett esophagus occurs in	Lower 1/3rd
Barrett esophagus can lead to	Stricture and peptic ulcer
Biopsy of Barrett’s esophagus every	2 years
Feature of Barrett’s esophagus	*Acidic and stains alcian blue*

CARCINOMA ESOPHAGUS

Predisposing factors of esophageal carcinoma	Plummer Winson syndrome, Tylosis Palmaris, Chronic achalasia
Esophageal adenocarcinoma predisposed by	Scleroderma, Barrett esophagus, Achalasia, Corrosive intake, Tylosis, Smoking, Lye stricture, Reflux esophagitis
Hyperkeratosis of palms and soles	Carcinoma esophagus
NOT a predisposing factor for Ca esophagus	Mediastinal fibrosis
NOT a predisposing factor for esophageal carcinoma	HPV
NOT true about Patterson Brown Kelly syndrome	LOWER esophageal web
Ellis classification	*Carcinoma esophagus*
MC type of Esophageal carcinoma in India	Squamous cell carcinoma
MC esophageal cancer world wide	Squamous cell carcinoma
Most prevalent esophageal carcinoma worldwide	Squamous cell carcinoma
Carcinoma esophagus in females is common in	Upper 1/3
MC type of Esophageal carcinoma in Upper one third	Squamous cell carcinoma
MC site of ca esophagus in India	Middle 1/3
MC Site of Ca Esophagus	Middle one third
MC site of carcinoma esophagus in India	Middle one third
MC site of squamous cell carcinoma esophagus	Middle one third
MC type of Esophageal carcinoma in Middle one third	Squamous cell carcinoma
MC site of adenocarcinoma esophagus	Lower 1/3
MC type of Esophageal carcinoma in Lower one third	Adenocarcinoma
Esophageal carcinoma	Common in men, Common in elderly, Adenocarcinoma is on rise, Dysphagia is most common symptom
NOT true about Ca esophagus	Pernicious anemia is often present

Filling defect & Rat Tail deformity	Ca Esophagus
Rat tail appearance of Barium swallow	Esophageal carcinoma
Most reliable diagnostic method for staging esophageal cancer	Endoscopic ultrasound
Early stage of carcinoma esophagus diagnosed by	Endoscopy
TNM staging of ca esophagus best done by	CT scan
On barium swallow, grade IV esophageal varies appear as	Mucosal folds below carina
Investigation for Carcinoma Esophagus	Endoscopy & Biopsy
Chemotherapeutic agent of choice for ca esophagus	Cisplatin
Neoadjuvant chemotherapy used in esophageal carcinoma	Cisplatin
MC side effect of cisplatin in patient of esophageal carcinoma	Acute tubular necrosis
Side effects of cisplatin	Nausea, Vomiting, Nephrotoxicity, Ototoxicity, Renal tubular damage, Secondary leukemia
Palliative treatment for esophageal carcinoma	Endoscopic LASER
Carcinoma resected earlier by mucosal resection	Ca Esophagus
Carcinoma which can be diagnosed early by mucosal resection	Esophageal carcinoma
Treatment of patient with carcinoma esophagus with malignant tracheal fistula	Palliative surgery (byepass)
Transhiatal approach for carcinoma oesophagus (Orringer)	Abdomen neck
Complication more in transthoracic esophagectomy than in transhiatal esophagectomy	*Pulmonary complication*
Iver Lewis Operation	Ca Esophagus, Subtotal Esophagectomy in Lower one third of Esophagus
Commonest cause of mortality in Iver Lewis operation	Anastomotic leak
Best substitute for esophagus after esophagectomy	Stomach

MALLORY WEISS SYNDROME

An old man presenting to emergency following a bout of prolonged vomiting with excessive hematemesis following alcohol ingestion	Mallory Weiss syndrome
Mallory Weiss syndrome	Seen in alcoholics, Partial thickness rupture occurs at gastric cardia, Patients who abuse analgesics, Pregnant females, Patients with hiatus hernia
Spontaneous rupture of esophagus	Mallory Weiss syndrome
Tear in Mallory Weiss syndrome	Mucosal
MC Site of tear in Mallory Weiss Syndrome	Immediately below Squamocolumnar junction at Cardia
Mallory Weiss tear occurs at	Gastroesophageal junction

HIATUS HERNIA

MC type of diaphragmatic hernia	Hiatus hernia

A 55 year male had retrosternal discomfort unrelated to physical exertion. Pain gets worse after lying down there is partial relief with antacids. most likely diagnosis is	Hiatus hernia
Sliding hernia	**Type I hiatus hernia**
Sliding hernia in all cases	Cardia goes through hiatus
Paraesophageal hiatus hernia	Surgery indicated in all asymptomatic cases, Paraesophageal type is more complicated
Paraesophageal hiatus hernia is also known as	*Volvolus of stomach*
Aim of preventing reflux esophagitis by repairing hiatus hernia is achieved by	Bringing stomach inferior to diaphragm, Reconstruction of angle of Hill, Repair of defect in diaphragm
MC complication of hiatus hernia	Esophagitis
Esophageal hiatus hernia is best diagnosed in	*Trendelenburg position*
Most useful investigation in sliding hernia in female	Barium meal
Hiatus hernia is treated by	Surgery when medical treatment has failed, Nissen's fundoplication
NOT an operation for Hiatus hernia	Heller's operation

GASTROESOPHAGEAL REFLUX DISORDER

Reflux esophagitis is prevented by	Long intraabdominal esophagus, Increased intraabdominal pressure, Right crus of diaphragm
Mechanisms preventing gastroesophageal reflux	Looping fibres of right crus of diaphragm, Mucosal folds at gastroesophageal junction, Angle made by esophagus with stomach
Most common mechanism of GERD	Transient decrease in pressure at lower esophagus
Burning pain on bending, lifting weights and lying down	Reflux esophagitis
Earliest indicator of GERD in infants	Respiratory symptoms
Connective tissue disorder associated with GERD	Scleroderma
Chest discomfort is due to acid reflux or not is tested by	**Bernstein test**
Bernstein test for	Pyrosis
Investigation of choice for GERD	24 hours pH study
Drug of choice for GERD	PPI
Cisapride useful in	GERD
Commonest side effect of cisapride	Diarrhea
Gastrokinetic drugs	Cisapride, domperidone
Female symptoms of flatulent dyspepsia. On proton pump inhibitors, controlled her symptoms. Next step	Wait and watch

ESOPHAGEAL PERFORATION

Commonest cause of esophageal perforation	Instrumentation
MC site of iatrogenic oesophageal perforation	Cervical portion
MC site of perforation of esophagus	Retrocricoid region
Area of esophagus mostly perforated during esophagoscopy	At cricopharyngeus muscle

Esophageal injury	Most common after penetrating injury, Can lead to mediastinal collection, Barium swallow is diagnostic, Treatment is primary repair, Mortality is increased if repair delayed beyond 24 hours
Perforation of oesophagus below instrumentation commonly occurs below	Cricopharyngeal sphincter
NOT seen in esophageal perforation	Bradycardia
In majority of patient with esophageal leak in thoracic cavity of less than 12 hours duration, the treatment of choice is	Primary closure, drainage and antibiotics

BOERRHAVE SYNDROME

MC site of spontaneous esophageal rupture	Distal end
Boerrhave syndrome is due to	Spontaneous perforation
Boerrhave disease shows perforation in	*Lower / posterior*
Meckler triad in Boerrhave syndrome	*Vomiting, lower chest pain and subcutaneous emphysema*
Boerrhave syndrome	Present with acute chest pain, Treatment is surgical

UPPER GI BLEED

Causes of upper GI bleed	Peptic ulcer, Erosive gastritis, Esophageal varices
MC cause of Upper GI bleeding in Children	Esophageal varices due to Portal Hypertension
MC cause of severe hematemesis in child	Portal hypertension
NSAID induced hematemesis is due to	*Gastric erosion*
Uncommon cause of hematemesis	Pernicious anemia, Ca stomach
Upper GI bleed associated with	Increased BUN
NOT true about GI bleeding	Angiography can image bleeding at a rate of 0.05 – 0.1 ml/min or less
NOT an indication for hospitalization in hematemesis	Hematocrit more than 40
Investigation of choice for upper GI bleed	Endoscopy
Most useful method to diagnose upper GI bleeding	Endoscopy
NOTES	**Natural Orifice Transluminal Endoscopic surgery**
In Forrest classification for bleeding peptic ulcer with a visible vessel or pigmented protuberance	F IIA
Most sensitive test to detect GI bleeding	Radiolabelled erythrocyte scanning
Least useful in diagnosis of acute hematemesis	Gastric content aspiration
Most appropriate therapy for upper GI bleed. mild splenomegaly, absence of other information	Intravenous pantoprazole
A patient presented to emergency ward with massive upper GI bleed. On examination he has mild splenomegaly in absence of any other information available, most appropriate treatment modality	Intravenous vasopressin
Following resuscitation, a patient with bleeding esophageal varices should be treated initially with	Sclerotherapy
A patient comes with hematemesis and malena, on upper GI scopy, no significant finding, 2 days later,	Laparotomy

patient rebleeds. Next line of investigation	

STOMACH AND SMALL INTESTINE

ANATOMY OF STOMACH

Blood supply of fundus of stomach	*Short gastric artery*
Nerve of latarjet is seen in	Stomach
Nerve of latarjet	*Anterior vagus*
Criminal nerve of Grassi	*Posterior vagus*
NOT a permanent mucosal fold	Gastric rugae
Neck cells are found in	Neck of gastric glands
Gastrin is mainly secreted by	Pyloric antrum
G cells are present mostly in	Pyloric antrum
Electrical pacemaker of stomach situated in	Fundus
Chief cells are seen in	Fundus
Part of gastric gland where chief cells are located	Fundus
Zymogen cells are abundant in	Fundus
Cardiopyloric mucous cells help in production of	*Glycoprotein*
Cells NOT found in stomach	Goblet cells
Most important blood supply to stomach	Left gastric artery
Blood supply of stomach	Left gastric artery, Short gastric artery, Left gastroepiploic artery

ANATOMY OF SMALL INTESTINE

Major duodenal papillae	*Situated 8 – 10 cm distal to pylorus on posteromedial wall*
Accessory pancreatic duct is situated	*2 cm above major duodenal papillae*
Small intestine	Zone of replication is in lower half of gland, M cells convey microorganism to Peyer patches
True about small intestine	GALT is in lamina propria, Close cell is NON secretory cells
Features of small intestine	*Mucous membrane has permanent folds called plicae circulares, no fatty tags attached to the wall, peyer's patches are found in small intestine, caliber of full small intestine is less than that of large intestine*
Cap is present in	*Proximal duodenum*
Brunner's gland are seen in	UPPER duodenum
Brunner's gland secrete	Mucus rich secretion
Cells found in small intestine	Stem cells, Goblet cells, Paneth cells
Cell which does NOT migrate from base of crypt to ends	Paneth cells

of villi	
NOT found in small intestine	Neck cells
Paneth cells	Rich in rough endoplasmic reticulum, High zinc content, Numerous lysozyme
Paneth cells contain	Zinc
Paneth cells secrete	*Defensin*
Paneth cells does NOT secrete	*Mucus*
NOT true about paneth cells	Foamy appearance
Supply first 2 cm of duodenum	Common hepatic artery, Gastroduodenal artery, Superior pancreaticoduodenal artery
Does NOT supply first 2 cm of duodenum	Supraduodenal artery
Valvulae coniventes are seen in	Jejunum, ileum
Dilated jejunal loops in Plain x ray abdomen	Valvulae connivente*s*
Feathery appearance in jejunum	Valvulae conniventes
Ileum	Lymphatic patch along antimesenteric border, LN in mesentry, 3-6 arcades in continuation, Smaller diameter than jejunum
Receptor for Vitamin B12 – IF complex in	Ileum
NOT true about ileum	Large circular mucosal folds
Contrast radiography	Ileum is featureless, Colon has haustrations, Jejunum is feathery, SUPERIOR part of duodenum has cap

GENERAL FEATURES OF STOMACH AND SMALL INTESTINE

Cause of stress ulcer	Burns, Cortisol therapy, Pulmonary insufficiency
Stress ulcer	Body and fundus
Stress ulcer after head injury	Cushing ulcer
Common sites of cushing ulcer	Proximal duodenum, Stomach, Oesophagus
NOT a common site of cushing's ulcer	Distal duodenum
Stress ulcer after burns	Curling ulcer
Curling ulcer	Shallow multiple ulcer, Seen in burn patients
MC site of Curling Ulcer	Duodenum
Site of Curling ulcer	First part of duodenum
Prepyloric or channel ulcer in stomach is termed as	Type 3
Canker sore	Aphthous ulcer
Transverse ulcers are seen in	Tuberculosis
Dieulafoy lesion	**Persistent caliber artery, most common site is lesser curvature**
Heterotopic gastric mucosa is also known as	**Esophageal inlet patch**
Drug induced gastritis is treated by	Omeprazole
Eosinophilic gastritis	Layer of presence in Gastric wall
Erosive gastritis commonly occurs at	Body
Chronic atrophic gastritis	**Type A – autoimmune, body predominant, type B – H.pylori, antral predominant**
Most likely to be associated with type A chronic gastritis resulting from autoimmune destruction of parietal cells	*Increased production of macrocytic red blood cells*
Histologic examination of lesion in stomach reveals fat laden cells. Likely cause is	Atrophic gastritis > Post gastrectomy

Watermelon stomach	**Gastric Antal Valvular Ectasia**
Treatment of Gastric antral vascular ectasia	Nd:Yag laser
Treatment of GAVE	Endoscopic LASER coagulation
Thickness of stomach in Ultrasound	5 mm
Thick mucous coat	Protective in ulcer patients
Function of thick gastric mucosa	Protects epithelium
Thickened gastric folds	Lymphoma, Menetrier's disease, Carcinoma, Eosinophilic gastritis
Hypertrophic gastropathy	Seen in Menetrier disease and ZES, Shows cerebriform rugosity in stomach, More in fundus and body
MC benign tumor of stomach	Adenoma
Double bubble sign	Duodenal atresia, Duodenal stenosis, Volvolus
Deficiency of abdominal muscle associated with	Eagle Barrette syndrome
MC site of regional enteritis	Distal ileum and colon
MC cause of small intestinal fistula	Postoperative
Most common symptom of Immunoproliferative small intestinal disease	Abdominal Pain
Duodenal adenocarcinoma	Most common small bowel carcinoma, Jaundice and anemia are common
Treatment of primary intestinal lymphangiectasia	*Low fat diet*
Anastomosis of small bowel is done by	Suture in 2 layers by absorbable suture
NOT an investigation for small intestine	USG enteroclysis
Kocher's maneuver	Mobilization of 2nd part of duodenum

ACUTE DILATATION OF STOMACH

Acute dilatation of stomach	Distension of stomach seen on X ray, Presents with vomiting, Aspiration, Atony of stomach
NOT true about acute dilatation of stomach	Resolves spontaneously without treatment
NOT a management of acute dilatation of stomach	Surgery

PEPTIC ULCER

Ulcer means	**Breach in mucosa > 5 mm and depth into submucosa**
MC cause of UGI bleed	Peptic ulcer
Peptic ulceration can occur in	Stomach, duodenum, Meckel's diverticulum
Peptic ulcer associated with	Cirrhosis, Zollinger ellison syndrome, Primary hyperparathyroidism, Smoking
Peptic ulcer NOT associated with	Pernicious anemia, Plummer Vinson syndrome
Peptic ulcer	Incidence of complication unchanged
Peptic ulcer	H.pylori
Intractable peptic ulcer with renal stones	Milk alkali syndrome
Common site of peptic ulcer	1st part of duodenum
MC Site of Peptic Ulcer	First Part of Duodenum, 2nd Antrum of Stomach, 3rd Gastroesophageal Junction
MC site of peptic ulcer perforation	Anterior aspect of first part of duodenum
MC cause of death in Peptic Ulcer	Perforation

MC cause of death in peptic ulcer patient	Hemorrhage
MC Complication of Acute & Chronic Peptic Ulcer	Hemorrhage
After 6-8 hours after peptic perforation the disappearance of abdominal wall rigidity is due to	Dilution of acid in the peritoneal cavity
Percentage of Patients with Perforated Peptic Ulcer Showing gas under Diaphragm	75%
Investigation of choice in peptic ulcer perforation	X ray abdomen
Treatment of perforated peptic ulcer	Iv fluids, Immediate surgery, Iv pantoprazole
Perforated ulcer may be treated by	Plication of perforation
Management of Perforated Peptic Ulcer	Onlay Omental Patch
Treatment of Peptic Ulcer Disease	Proton Pump Inhibitor
PPIs for peptic ulcer should be taken in	Before breakfast
Despite having short half-life, PPI acts longer because	Causes irreversible inhibition of proton pump. Acid secretion suppressed till new pumps are generated
Peptic disease	Misoprostol is DOC in patients on NSAID, DU is prevented by use of single night time H2 blockers, omeprazole may help ulcers refractory to H2 blockers
Beneficial in NSAID induced peptic ulcer	PGE1 agonist
Anticholinergic specially used in peptic ulcer	Pirenzepine
Anti peptic ulcer drug that can be given in patients with chronic renal failure	Sucralfate
Sucralfate interfere with absorption when given with	Phenytoin, Digoxin, Ciprofloxacin
Ranitidine and sucralfate is incorrect combination because	Sucralfate inhibits absorption of ranitidine
Cimetidine cause	Decrease in sperm count, pepsin secretion is NOT affected, Gynacomastia, Potent H2 blocker
Effectiveness of antacid at pH	3,5-15 min
Commonest operation for peptic ulcer with gastric outlet obstruction	Truncal vagotomy with gastrojejunostomy
Highest risk of recurrence of peptic ulcer occur after	Duodenojejunostomy
Lowest recurrence of peptic ulcer associated with	Vagotomy and antrectomy

H.PYLORI

Urease producing organisms	H.Pylori, Proteus, Klebsiella
Bacterial genome completely recognized for	H.pylori
H.pylori	50% of world population affected, 85% of population affected in developing countries, infection is common in low socioeconomic status, urease activity provides protective environment to bacilli, gram negative flagellate, urease breath test positive, risk factor for adenocarcinoma of stomach
H.pylori	With chronic infection urease breath test does NOT become negative, H.pylori infection remain lifelong if untreated, Endoscopic is diagnostic, Toxicogenic strain usually cause ulcer, Gram negative organism, More prevalent in developing countries
H.pylori	**cagA+**
H.pylori causes carcinoma by	*cagA, vacA and elaboration of IL-8*
H.pylori infections are commonly seen in	*Low socioeconomic status*

H.pylori	Obligate parasite on gastric mucosa
Growth factor required by H.pylori	Both V and X
H.pylori causes carcinoma by	Gastric metaplasia
H.pylori causes	Chronic atrophic gastritis
H.pylori causes	Gastric ulcer, duodenal ulcer, chronic atrophic gastritis
Patient with H.pylori infection show a blood picture of	Iron deficiency anemia
H.pylori known to cause	Gastric ulcer, duodenal ulcer, gastric lymphomas
H.pylori DO NOT invade	Gastric mucosa
H.pylori gastritis involves	Antrum
Gastritis caused by H.pylori	Type B
H.pylori cause carcinoma by	Intestinal metaplasia
NOT true about H.pylori associated gastritis	Intraepithelial neutrophils
MC Site of Involvement of MALT Chronic H.pylori Infection	Antrum
MALT	Present in extranodal sites, Predisposed by H.pylori infection, Sensitive to chemotherapy, Stromal polyp infrequent
H.pylori eradication is NOT of benefit in	Hypertrophic gastritis
NOT a feature of H.pylori	Fundal atrophic gastritis
Helicobacter pylori NOT associated with	Gastric leiomyoma
H.Pylori is NOT associated with	Gastric adenoma
Diagnostic tests for H.pylori	Urea breath test, rapid urease test, gastric biopsy and Warthin starry stain
Best method to test presence of residual H.pylori infection	Urease breath test
Test of choice for eradication of H.pylori	Urease breath test
H.pylori is stained with	Sterner silver
Endoscopic biopsy from a case of H.pylori related duodenal ulcer most likely to reveal	Antral predominant gastritis
NOT a diagnostic test for H.pylori	SAFA
Bismuth subcitrate is used for	H.pylori eradication, gastric ulcer protection, duodenal ulcer healing
Drug regimen against H.pylori	Amoxicillin, Bismuth subcitrate, omeprazole
Drugs used for H.pylori	Metronidazole, Omeprazole, Amoxicillin, Bismuth
NOT effective against H.pylori	Erythromycin, Mosapride
Drug NOT commonly used in H.pylori	Oxytetracycline, cisapride
Epidemiological studies of H.pylori done by	Serological markers
Eradication of helicobacter pylori has been proved be beneficial in	Low grade malt lymphoma
Translocation associated with MALT lymphoma	**(11,18)**
Treatment of MALToma	**Chlorambucil**
Duration of antibiotic therapy for H.pylori peptic ulcer	**2 weeks**

GASTRIC ULCER

Incidence of H.pylori in gastric ulcer	60%
MC cause of chronic gastric ulcer	Massive hematemesis
Classification of Modified Johnson classification	*Gastric ulcer*
Types of gastric ulcer	Type I – fundus, type II – antrum, type III – within 3 cm

	of pylorus, type IV - cardia
MC Site of benign Gastric Ulcer	Lesser Curvature near Incisura angularis (Junction of body and antrum)
Gastric ulcers are caused due to	Decreased mucosal resistance
Leather bottle deformity in stomach	Gastric ulcer
Hour glass stomach	Gastric ulcer
An old hypertensive man, on treatment with Aspirin, Sorbitrate, and Atenolol suddenly fell in bathroom. Relatives noted black stools. He presented in ER with hypotension, tachycardia. The diagnosis is	Gastric Ulcer
Malignant transformation is commonly seen in	Chronic gastric ulcer
Gastric ulcer associated with	Normal or DECREASED gastric output
Pentagastrin fast achlorhydria in patient with gastric ulcer	Malignant ulcer
Features of malignant gastric ulcer on barium meal	Location on greater curvature, carman's meniscus sign, radiating folds which do not reach the edge of ulcer
Posterior Perforating ulcer of Pyloric antrum is most likely to perforate	Lesser Sac/Omental Bursa
Antiemetic also decreases acid secretion due to its action on H1 receptor	Promethazine
NOT a H2 blocker	Omeprazole
Proton pump inhibitors are most effective when given	Shortly before meals
Proton pump inhibitor having enzyme inhibitory activity	Omeprazole
Esomeprazole acts by inhibiting	H+K+ ATPase
Inhibition of HCL secretion by omeprazole occurs within hour, reaches peak at 2 hours, plateau by 4th day. After how many days, will the secretion gradually normalize	3-5 days
Treatment of Proton Pump inhibitor	Omeprazole
PPI containing imidazopyridine ring	**Tenatoprazole**
Agents beneficial in NSAIDs induced gastric ulcer	PGE1 agonist
Ranitidine and sucralfate prescribed in morning hours, this is incorrect because	Ranitidine increases the gastric pH so sucralfate not able to act
Patient taking famotidine, sucralfate and antacid. Treatment is irrational because	Sucralfate polymerises only when gastric pH less than 4
Sucralfate polymerises only when	Gastric pH less than 4
Affect the integrity of adherent gel of sucralfate interfering with its action	Antacid
Only non particulate antacid	*Sodium citrate*
Rationale behind combination of magnesium and aluminium in antacid	*Magnesium tends to cause laxative effect that counteracts the tendency of an aluminium salt to cause constipation*
NOT an antacid	Magnesium sulphate
Ulcer protecting agent	Colloidal Bismuth subcitrate
Side effects of bismuth	**Black stools, blackening of tongue**
Surgical treatment of type I gastric ulcer	*Distal gastrectomy followed with a Billroth I gastrectomy*

DUODENAL ULCER

Factors contributing to development of duodenal ulcer	Alcohol, Gastric acid, Smoking
May contribute to development of duodenal ulcer	I 131
NOT a precancerous condition	Duodenal ulcer
Duodenal ulcer	Infection with H.pylori and NSAID induced injury account for majority of duodenal ulcer, Malignant duodenal ulcers are extremely rare, Eradication of H.pylori has greatly reduced the recurrence rate in duodenal ulcer
42 year old company executive, sudden upper GI bleed of bright red colour, no significant previous history	Duodenal ulcer
65 year patient with coronary artery disease on aspirin for 2 years. Complains of black stools. abdominal examination is normal	Duodenal ulcer
2*2 ulcer on posterior aspect of first part of duodenum	Bleeding vessel seen could NOT be controlled endoscopically
Source of bleeding in duodenal ulcer	Gastroduodenal artery
MC Source of Hemorrhage in Duodenal Ulcer (Arterial)	Gastroduodenal Artery
MC cause of Generalized Peritonitis in 40 years old adult male	Duodenal Ulcer Perforation
Complication commonly occurring in anterior duodenal ulcer	Perforation
Prognosis in duodenal ulcer	Age, Duration of history, Peritonitis
Endoscopy is NOT useful in diagnosis of peptic ulcer in	Giant duodenal ulcer
Single dose H2 blocker in treating duodenal ulcer is given at	Bedtime
Primary aim in duodenal ulcer management	Prevention of complications
NOT an indication of surgery in a case of duodenal ulcer	Typical periodicity
Graham's patch is used in	Duodenal ulcer closure
Most common operative procedure for perforated duodenal ulcer	Graham's omentum patch repair
Treatment of Choice for Chronic Duodenal Ulcer	Highly Selective Vagotomy
Treatment of Choice for RECURRENT Chronic Duodenal Ulcer	Vagotomy with Antrectomy
Recurrent duodenal ulcer 2.5 cm procedure of choice	Truncal vagotomy with antrectomy
When vagotomy is done for duodenal ulcer, it is important to	Add drainage procedure, perform Hollander test pre and postoperatively, retain vagal fibres to liver and pancreas
Least recurrence rate in duodenal ulcer treatment is seen with	Truncal vagotomy with antrectomy
Lesser Curvature anterior Seromyotomy done for	Duodenal Ulcer
Prostaglandin used in prevention of duodenal ulcer	Misoprostol

DUODENAL STRICTURE

Duodenal strictures caused by	TB, Ca pancreas, Crohn's disease
Best line of management of duodenal stricture	Duodenojejunostomy

DUMPING SYNDROME AND VAGOTOMY

Early dumping is aggravated by	*More food*
More food relieves	*Late dumping*
Dumping syndrome is due to	Hyperosmolar contents in small intestine
Dumping syndrome is least likely to occur in	Highly Selective vagotomy
Recurrence is least with	Truncal vagotomy and antrectomy
In highly selective vagotomy, vagal supply is severed to	Antrum
Least operative mortality in duodenal ulcer surgery in	*Highly selective vagotomy*
Factors responsible for causing diarrhea after vagotomy	Rapid gastric emptying, Hypoacidity in duodenum, Irregular peristalsis
Treatment of late dumping	*Octreotide*
Post vagotomy diarrhea can be effectively managed by	Somatostatin analogue
Maximal reduction in gastric acidity is achieved by	Truncal vagotomy with antrectomy
Commonest early complication of truncal vagotomy and gastrojejunostomy	Anastomotic hemorrhage

POST CIBAL SYNDROME

Diarrhoea cramps postprandially, CVS finding	Postcibal syndrome
Post cibal syndrome	Managed conservatively, Hypermotility of intestine is common
NOT true about early postcibal syndrome	Surgery is indicated

GASTRIC OUTLET OBSTRUCTION

MC cause of Metabolic alkalosis in surgical patient	Gastric outlet obstruction
MC cause of Gastric outlet obstruction In India	Cancer stomach
MC Site of Gastric outlet Obstruction	Duodenum
In gastric outlet obstruction in a peptic ulcer patient, site of obstruction is most likely to be	Duodenum
35 year male, had chronic duodenal ulcer for last 6 years presents with worsening of symptoms loss of periodicity of symptoms, pain on rising in morning sense of epigastric bloating and post prandial vomiting. most likely cause of worsening of symptoms is development of	Gastric outlet obstruction
Most severe degree of alkalosis occur in obstruction of	Pylorus
NOT a complication of trichobezar	Malignancy
OPD management of Gastric Outlet Obstruction	Normal Saline + Potassium
Fluid management of GOO in OPD	Normal saline, Normal saline with potassium

MENETRIER DISEASE

Menetrier Disease	Enlarged Gastric Rugae , Protein Losing Enteropathy, Premalignant condition, Gastric acid usually absent
NOT true about Menetrier disease	Exophytic growth

FEATURES OF CARCINOMA STOMACH

Blood groups associated with gastric carcinoma	Blood group A
Precancerous condition of ca stomach	Chronic gastric atrophy, Pernicious anemia
Forerunner of carcinoma stomach	*Chronic gastric ulcer*
Most significant risk factor for development of gastric carcinoma	Intestinal metaplasia
Premalignant conditions of Gastric carcinoma	Atrophic gastritis
Risk factor for development of gastric carcinoma	Intestinal metaplasia type III
Do NOT predispose to carcinoma stomach	Gastric erosion
NOT a risk factor for Carcinoma Stomach	Decreased salt intake
NOT a premalignant condition for gastric carcinoma	Hiatus hernia
NOT having malignant potential	Hyperplastic polyp, Duodenal peptic ulcer
Does NOT predispose to ca stomach	Gastric erosion
NOT a predisposing factor for gastric cancer	O blood group
MC site of gastric cancer	*Gastroesophageal junction*
Ca stomach	Weightlessness is common feature, Radioresistant
Early gastric carcinoma	Invasion of mucosa and submucosa irrespective of lymph node spread, Radical gastrectomy preferred, Endoscopic removal of lesion
Early gastric carcinoma	Gastric adenocarcinoma confined to mucosa and submucosa
Early gastric cancer	*I – protruding, IIa – elevated, IIb – flat, IIc – depressed, III - excavated*
MC advanced gastric carcinoma	*Ulcerative*
MC site of ca stomach	Proximal stomach
MC site of carcinoma stomach	Prepyloric
MC Site of Gastric Carcinoma	Antrum, Lesser curvature > Fundus
MC Site of Gastric Adenocarcinoma secondary to H.Pylori	Antrum
Gastric carcinoma occurring secondary to pernicious anaemia is in	Fundus
MC Presentation of Ca Stomach	Weight Loss
MC site of stomach malignancy	Adenocarcinoma
Sister Mary Joseph nodule is commonly seen with	Stomach cancer
Seborrhoeic keratosis in gastric adenocarcinoma	**Leser Trelat sign**
Carman meniscus sign is seen in	*Carcinoma stomach*
Irish node is pathognomic of	Ca stomach
50 year old woman, bilateral solid ovarian tumours, ascites, ulcerative growth in pyloric region of stomach	Carcinoma stomach with Krukenberg tumour
Gastric carcinoma often associated with	Hypochlorhydria/achlorhydria
Linitis plastica	Ca stomach

MC site of leiomyosarcoma in GIT	*Stomach*
Most hemorrhagic tumor	Leiomyosarcoma stomach
Troisier sign	Left supraclavicular lymphnode enlargement
Mucinous ascites	Stomach carcinoma
Tumour does NOT cause bony metastasis	Gastric carcinoma
Hypercalcemia is NOT seen in	Gastric carcinoma
NOT a feature of carcinoma of body of stomach	Dysphagia
NOT true about gastric cancer	Incidence increasing worldwide
NOT a feature of silent carcinoma of body of stomach	Dysphagia
Best prognosis in carcinoma stomach	Superficial spreading type
Proliferative gastric carcinoma	*Good prognosis*

MANAGEMENT OF CARCINOMA STOMACH

Schirrhous carcinoma of stomach is best diagnosed by	Barium study
Early mucosal cancers of stomach can be better made out by	Double contrast
Early Diagnosis of Gastric cancer	Staining with Endoscopic Biopsy
Most sensitive test for detecting T stage of gastric cancer	Endoscopic ultrasound
Management of carcinoma stomach	*ECF chemotherapy*
NOT an indication of operability in ca stomach	Krukenberg tumor
Krukenberg tumor is NOT seen in	*Postmenopausal woman*
Physiological gastrectomy	Ligating all major arteries
Treatment of choice for gastric cancer	Surgery
Distal gastric tumour and direct invasion to distal pancreas	Partial gastrectomy and distal pancreatectomy
Partial gastrectomy patient presenting with neurological symptoms	Vitamin B12 deficiency
A 60 year old male, carcinoma stomach, CT scan showed a mass measuring 4*4 cm in antrum with involvement of celiac nodes and right gastric nodes. management of choice	Subtotal gastrectomy
Gastric Cancer – Standard Operation	Radical Subtotal Gastrectomy
Csende procedure	**Subtotal gastrectomy with roux en y esophagejejunostomy**
First gastrectomy was performed in 1881 by	Billroth
Venting gastrostomy for	**Palliation**
If the stump of stomach is directly anastomosed to stump of duodenum is called	Billroth I gastrectomy
Anemia is common in	Billroth II
Earliest manifestation seen after gastrectomy	Loss of storage capacity
Ideal treatment for alkaline reflux gastritis after billroth I and billroth II	*Roux en Y gastrojejunostomy*
Duodenal blow out following gastrectomy most commonly occurs on	4th day
Duodenal blow out	Complication of partial gastrectomy
Does NOT occur after gastrectomy	Fluid loss

HYPERTROPHIC PYLORIC STENOSIS

Drug that can cause pyloric stenosis	Erythromycin
Congenital Hypertrophic Pyloric Stenosis	First born male child
Congenital hypertrophic pyloric stenosis is due to	Hypertrophy of longitudinal muscles of pylorus
In congenital pyloric stenosis, defect usually in	Circular fibres of antrum
Robust male baby, vigorous feeding and immediate vomiting at 2 months of age. most probable diagnosis	Congenital hypertrophic pyloric stenosis
Congenital hypertrophic pyloric stenosis	Non bilious vomiting, Projectile vomiting, Non projectile vomiting, Forceful
Congenital hypertrophic pyloric stenosis present after	One month
Most essential feature in reaching a diagnosis of hypertrophic pyloric stenosis	Palpation of hypertrophic pylorus
Congenital hypertrophic pyloric stenosis usually presents	Around 2 weeks after birth
Olive shaped mass	Pyloric stenosis
Characteristic clinical feature of congenital hypertrophic pyloric stenosis	Palpable mass
Characteristic of congenital hypertrophic pyloric stenosis	Pyloric tumour best felt during feeding
Projectile vomiting is a feature of	Hypertrophic pyloric stenosis
Rabbit pellet stools	*Congenital hypertrophic pyloric stenosis*
NOT a clinical feature of congenital hypertrophic pyloric stenosis	Bilious vomiting
NOT a cause of vomiting on first day of baby's life	Pyloric stenosis
Hypertrophic pyloric stenosis is NOT characterized by	Alkaline urine
MC Biochemical abnormality in Congenital Hypertrophic Pyloric Stenosis	Hypokalemic Hypochloremic Metabolic alkalosis with Paradoxical aciduria
Feature of Congenital hypertrophic pyloric stenosis	Hyperchloremic alkalosis
NOT a feature of Congenital hypertrophic pyloric stenosis	Hypocalcemia
NOT true about congenital hypertrophic pyloric stenosis	Diarrhea
Diagnostic test for Pyloric Stenosis	USG
String Sign	Hypertrophic Pyloric Stenosis
Shoulder sign, double track sign	*Hypertrophic pyloric stenosis*
Pyloric canal in Congenital Hypertrophic Pyloric Stenosis	>16 mm
NOT an USG finding of congenital pyloric stenosis	High gastric residue
Neonate posted for pyloric stenosis surgery. Reduced serum calcium. Investigation before administering calcium	Serum protein
3 week old child presenting with vomiting and failure to thrive is found to have pyloric stenosis. next step of management	Correction of electrolyte disturbance
Treatment of metabolic derangement in congenital hypertrophic pyloric stenosis	*2.5% dextrose + 0.45% NaCl + 1 gram of KCl per 500 ml of fluid*
Treatment of choice in congenital hypertrophic pyloric stenosis	Pyloroplasty
Fredet Ramsted Pyloromyotomy	Hypertrophic Pyloric Stenosis
If the mucosa was accidentally opened at operation (Ramsted) it is wise not to feed the child orally for	24 hours

POLYPOSIS

Familial adenomatous polyposis	Autosomal dominant, Screening by sigmoidoscopy, Polyps develop in early adulthood, Epidermal cysts and osteomas may occur
Familial polyposis coli	Increased expression of c myc gene
Familial polyposis coli associated with	Cancer
Following genetic counseling in a family for familial polyposis coli, next screening test	APC gene
APC gene	*Chromosome 5q*
Significant risk factors for colonic carcinoma in an adenomatous polyp	Sessile polyp, Villous histology, Size > 2 cm, Atypia
Highest malignant potential	Familial polyposis
Polyp having highest risk of progression to carcinoma	*Adenomatous polyp*
FAP	*Left sided*
HNPCC	*Right sided*
NOT true about FAP	Autosomal recessive
Incidence of malignancy is maximum in	Villous adenoma
Polyp having maximum risk of turning into malignancy	Tubular villous adenoma
Villous polyp of rectum	Mucus diarrhea with hypokalemia
Lost in villous papilloma of rectum	K+
Metabolic abnormality in large colorectal villous adenoma	Hypokalemic metabolic acidosis
Best surgical management of villous adenoma of rectum	Local resection of lesion
Malignant potential of colonic polyp	Polyps of familial polyposis coli and villous adenoma high risk
NOT true about malignant potential of colorectal polyp	Pseudopolyps of ulcerative colitis has high risk of malignancy
Neoplastic colorectal polyp	Sessile polyps > 1 cm is malignant, MC site is colon and rectum, Adenomatous polyp is premalignant
Turcot syndrome	FAP , Brain tumors
Gardener syndrome	Polyposis colon, osteoma, epidural inclusion body, fibrous tumour of skin
MC facial anomaly in Gardner syndrome	Multiple osteoma
Gardner syndrome	Polyposis colon, osteoma, epidermal inclusion cyst and fibrous tumors of skin
NOT true about gardener syndrome	Always in 5th decade of life
Commonest gastric polyp	Hyperplastic polyp
MC type of polyp in children	Juvenile polyp
NOT premalignant	Juvenile polyp
Congenital abnormalities associated with juvenile polyp	*Malrotation, meckel's diverticulum, mesenteric lymphangioma*
NOT premalignant	Ulcerative colitis, Hyperplastic polyp, Juvenile polyp
Polyp having maximum malignant potential	Sessile
On colonoscopy, which is highly malignant	Multiple flat polyp about hundreds

GASTRIC DIVERTICULA

Diverticulum of stomach	Pain is the main symptom, Usually at cardiac end, Usually on posterior surface
Most frequent symptom of gastric diverticula	Epigastric pain

GASTRIC FISTULA

A pt undergone gastrojejunostomy suddenly develop severe diarrhea. which should be suspected	Gastrojejunocolic fistula
Best method of treatment for gastrojejunocolic fistula	Resection of stomach, jejunum and colon
Highest pickup of gastrojejunocolic fistula by	Ba enema

GASTROINTESTINAL STROMAL MALIGNANCY

c-kit oncogene is associated with	**GIST**
Gastrointestinal stromal malignancy arise from	Interstitial cells of Cajal
GIST is most common in	Stomach
Interstitial cells of cajal are found between	*Longitudinal and circular smooth muscle fibers*
NOT true about GIST	More common in duodenum
Investigation of choice for recurrent GIST	PET CT
Imatinib is used in treatment of	GIST
Imatinib acts by inhibition of	Tyrosine kinase
Imatinib	Tyrosine kinase inhibitor, Competitive inhibition of ATP binding site of abl kinase
Mechanism of action of imatinib mesylate	Blocks action of chimeric fusion protein of bcr-abl
Duration of imatinib therapy in advanced GIST	*Until progression or intolerance*
Side effect of imatinib	*Pedal edema*
Drug approved for imatinib resistant GIST	**Sunitinib**
New drug for GIST	*Regorafenib*
Indication for surgery in gastric lymphoma	Bleeding, Perforation, Residual disease following chemotherapy
NOT true about gastric lymphoma	Total gastrectomy with adjuvant chemotherapy is the treatment of choice
NOT an indication for surgery in gastric lymphoma	Intractable pain

SMALL INTESTINE TUMOURS

Commonest tumor of small intestine	Leiomyoma
Small intestine tumor	Palliative surgeries are done even in presence of metastasis, Commonest site terminal ileum, Chemotherapy is of no proven significance
True regarding benign small bowel tumors	Accidently discovered in surgery

Duodenal adenocarcinoma	Arises from periampullary carcinoma, Jaundice and anemia found, Surgery is only curative
Lipoma common in intestine in	Ileum
Duodenal somatostatinoma is associated with	*Von Recklinghausen syndrome*
Investigation of choice for small intestine tumor	CT with contrast
Investigation of choice for small intestinal tumor	Ba meal follow through
NOT an investigation for small intestine abnormalities	USG enteroclysis
Small intestinal biopsy is diagnostic in	Whipple disease, Abetalipoproteinemia, Agammaglobulinemia

PEUTZ JEGHER POLYPOSIS

Peutz Jegher Polyposis	LKB1
Malena, hyperpigmentation over lips, oral mucosa, skin. Sister is also having similar complaints	Peutz jegher syndrome
Pigmentation of lip, oral mucosa and intestinal polyp. sister gave same history	Peutz Jegher polyposis
Pigmented macules over palm, sole and oral mucosa, anemia and pain in abdomen	Peutz jegher syndrome
MC Site of Peutz Jegher Polyposis	Proximal Jejunum
Characteristic skin lesion in Peutz Jegher syndrome	*Lentigenes*
Oral examination is done in case of	Peutz jegher syndrome
Polyps In Peutz Jegher syndrome	Small bowel
Least malignant potential	Hamartomatous polyp in peutz Jegher syndrome

LARGE INTESTINE

GENERAL FEATURES OF LARGE INTESTINE

Large intestine	Secretes ALKALINE mucous which helps in formation of stools, Site of mucocutaneous junction, Its epithelium contains goblet cells in large numbers, Absorbs salt and water
Griffith point in	**Splenic flexure**
Sudeck point	**Sigmoid colon**
Differentiating X ray feature between large and small intestine	Feces
Colonic organism	10^10 to 10^11 organisms
In the gut, anaerobic bacteria outnumber aerobic bacteria by	1000:1
In intraepithelial region of mucosa of intestine the predominant cell population is that of	T cell
NOT important for maintenance of normal fecal incontinence	Haustral valve
Antiperistalsis seen in	Distal colon

Part of large intestine with least diameter	Ascending colon
Area of colon least visualized by barium studies	Sigmoid
Tenesmus is seen in lesion of	Sigmoid colon
Appendices epiploicae present in	Sigmoid colon
Blood supply to sigmoid colon	Marginal artery, Sigmoid artery
Flexible sigmoidoscope	*60 cm*
Curvatures of rectum	*Upper and lower rectum – convex to right, middle – convex to left*
Houston valve	*Rectum*
Valves of Houston	They are more prominent on distension, Anteriorly correlate with peritoneal reflection, Are placed transversely, Contains all layers of intestine
NOT true about valves of Houston	Disappear on distension
Length of proctoscope	4 inches
Proctoscope can be used to inspect lower rectum up to	10 cm
Terminal group of lymphnode to colon	Preaortic
Epicolic node	Node draining colon
Polyp most commonly seen in rectum	*Hyperplastic polyp*
Investigation of choice to detect small paraaortic lymph nodes	Lymphangiography
Lymphangiography finding of secondary nodes	Irregular pattern
Residual lymphnodes after resection of lymphoma diagnosed by	Bipedal lymphangiography
Most common cause of acute mesenteric ischemia	Embolism
60 years male, IHD and atherosclerosis, abdominal pain and maroon stools	Acute mesenteric ischemia
Duplication of intestine	Spherical type is most common, Tubular type is attached longitudinally with bowel
Duplication of GIT commonly involves	Ileum
Commonest site of ischemic colitis	Splenic flexure
Thumb printing is characteristic of	Ischemic colitis
A newborn baby presents with absent anal orifice and meconuria. Appropriate management	Transverse colitis
A patient with leukemia on chemotherapy develops acute right lower abdominal pain associated with anemia, thrombocytopenia, leukopenia	Neutropenic colitis
Telangiectasia of colon	Age group less than 40 years and more than 60 years, Common site is caecum, Rectum very less involvement
Commonest benign tumour of large bowel	Polyp
Functional GI disorders differentiated from organic disorders by	Bleeding per rectum
A patient suffered bullet injury to left side of colon and presented in casualty after 12 hours	Proximal colostomy and bridging out the distal end as mucus fistula
GI disorder predisposing to urolithiasis	Short bowel syndrome
Gangrene of intestine NOT associated with	Tricuspid valve endocarditis
Anus vestibularis is a type of	Vulvovaginal anus
Melanoma of anal canal	Present usually as bleeding

LOWER GI BLEEDING

MC cause of rectal bleeding in infancy	**Fissure**
MC cause of lower GI bleed in India	Hemorrhoids
Minimum amount of GI bleed to cause malena	60 ml
NOT a cause of blood in stools in children	Carcinoma
Most useful investigation for profuse lower intestinal bleeding	Colonoscopy
Investigation of choice in elderly with occult blood in stool and anemia	Colonoscopy

FEATURES OF CARCINOMA COLON

Most protective against carcinoma colon	High fibre diet
Chromosome involved in progression of adenoma to carcinoma	*Chromosome 17p*
MC type of inherited carcinoma colon	*Hereditary non polyposis colonic cancer*
Strong correlation with colorectal carcinoma	Familial polyposis coli
Dietary factors associated with carcinoma colon	Low fibre, High fat intake, Japanese more prone to develop ca colon, Animal fat consumption
Risk factors for ca colon	FAP, Ulcerative colitis, Crohn’s disease
Highly refined carbohydrates cause carcinoma of	Colon
Most precancerous condition for carcinoma colon	Familial polyposis
Strongest correlation with colorectal cancer	Familial polyposis
Colonic cancer NOT associated with	Rb
NOT a risk factor for colonic carcinoma in adenomatous polyp	Peduculated polyp
Not a mutation in colon ca	C-MYC
Gene NOT involved in development of carcinoma colon	Beta catenin
NOT a precancerous condition of colon	Irritable bowel syndrome
Multistep carcinogenesis best seen in	Cancer colon
MC Site of Ca Colon	Rectosigmoid
Large percentage of carcinoma colon are found in	Descending and sigmoid colon
Full bowel preparation is MUST in	Carcinoma colon
MC Type of Ca Colon	Adenocarcinoma
MC Type of Ca Right Colon	Fungating
Cauliflower like growth is common in	*Ascending colon*
MC Type of Ca Left Colon	Stenosing
Ca colon	1/3rd cases show liver metastasis, Obstruction common in sigmoid colon, CEA is useful for prognostic importance, Duke staging, Alternate bowel habit is common in ca left colon
Ca colon	Right sided ca colon has better prognosis than left sided ca colon
Carcinoma right colon presents as	Anemia, Mass in right iliac fossa
Carcinoma left colon present as	Obstruction, Altered bowel habit
Hyperechoic Hypervascular Hepatic Metastasis on USG	Colorectal Carcinoma (Mucinous Adenocarcinoma)
Apple core sign/napkin ring sign	Carcinoma colon

Duke's classification for	Colorectal carcinoma

DIAGNOSIS OF CARCINOMA COLON

Cancer in which overall survival increased by screening procedures	Ca Colon
CEA raised in	Ca colon
Screening for occult blood is useful in	Ca colon
Stool positive for occult blood, next investigation	Colonoscopy
Guiac test	Occult blood in stool
Radiological investigation of choice for Ca Colon	Double Contrast Barium enema
Best investigation for colonic carcinoma	Colonoscopy and biopsy
Yeoman forceps for biopsy of	*Carcinoma rectum*
Tendency of colonic carcinoma to metastasize is best assessed by	Depth of penetration of bowel wall
Single most Prognostic factor for Colorectal Carcinoma	Stage of Disease
Single most important independent factor for Colorectal Carcinoma	Lymphnode status
Absolute contraindication for Barium enema	Toxic Megacolon

TREATMENT OF CARCINOMA COLON

Complete bowel preparation is done for	Colonic carcinoma
Folfox regimen for	**Colorectal carcinoma**
Adjuvant chemotherapy is of definite value in	Carcinoma colon
In case of elective surgery for ca sigmoid colon	Broad spectrum antibiotic at the time of operation
Immunoguided surgery done for	Ca Colon
A patient was operated for colonic carcinoma and later a solitary nodule was found in the liver, treatment of choice	Surgery
After undergoing surgery for carcinoma colon, single liver metastasis of 2 cm. next step	Resection
Treatment of unresectable liver metastasis	*Folinic acid and 5 fluorouracil*
Treatment of ca left colon with acute obstruction	Primary resection with Hartman procedure, Defunctioning colostomy, Resection of whole left bowel and end to end anastomosis, Left colectomy with anastomosis, Proximal colostomy, Extended right colectomy with ileoanal anastomosis
A 60 year old male with ca descending colon with acute intestinal obstruction. in emergency department treatment of choice	Hartman procedure
Ca colon	Solitary liver metastasis is NOT a contraindication for surgery
Appearance of anastomotic leakage following a low colonic anastomosis most often manifests	5-10 days
Most likely cause for suture leak after colon resection and anastomosis for cancer sigmoid	Ischemia
After hemicolectomy, on 6th postoperative day patient	Start IVF, Urgent laparotomy

developed serous discharge from wound.	
Enterocutaneous fistula	High output fistula drains 500 ml/day, Fluid and electrolyte loss can occur
MC cause of enterocutaneous fistula	*Previous surgery*

FEATURES OF CARCINOMA RECTUM

Rectal carcinoma	Most common adenocarcinoma, Surgery is the treatment of choice, Surgical treatment indicated in spite of hepatic metastasis
MC Type of Rectal Carcinoma	Adenocarcinoma
Most Aggressive Rectal Carcinoma	Secondary Mucoid Carcinoma
MC Presentation of Ca Rectum	Bleeding Per Rectum
NOT an early feature of Ca rectum	Pain
Acquired megacolon is seen in	Rectal malignancy
Anterior resection is the treatment of choice for	Ca rectum
Prognosis of carcinoma rectum is best assessed by	Histological grading

MANAGEMENT OF CARCINOMA RECTUM

Assessment of depth of penetration of perirectal nodes in rectal cancer	MRI
A punch biopsy shows carcinoma rectum with fixed mass. Chest X ray normal. Least useful investigation	Rigid proctoscope
Chemotherapy for carcinoma rectum	*5 fluorouracil, folinic acid*
Treatment of Choice for Ca Rectum	Surgery
Best procedure in mid rectal carcinoma	Anterior resection
Management of carcinoma near anorectal junction	Abdominoperineal resection
Pelvic exenteration	Brunshwing's operation
Management of Rectal carcinoma	Hartmann procedure
Unaffected tissue margin resected in carcinoma rectum	*1 cm*
Length of anal canal	4 cm
Distal clearance in surgery for ca rectum	2 cm
In carcinoma of anus, distal margin of clearance of anal canal of at least	2 cm
3.5 cm above anal verge	Abdominoperineal resection
Rectal carcinoma 5 cm from anal verge	Abdominoperineal resection
7 cm above anal verge	Anterior resection
6 cm from dentate line	Anterior resection
Treatment of Ca rectum 12 cm from anal verge	Anterior resection
APR is done in colorectal carcinoma on the basis of	Distance from anal verge

CARCINOMA ANAL CANAL

NOT an anal margin tumour	Melanoma

Commonest type of anal canal carcinoma	Squamous cell (epidermoid)
Metastasis in ca anus	Inguinal lymphnode
Treatment of squamous cell carcinoma anus	Chemoradiation
Treatment of squamous cell carcinoma of anal canal	Cisplatin based chemotherapy followed by radical radiation
Nigro regimen	Regimen for anal canal neoplasm, Incorporates chemotherapy with radiation as an alternate to surgery, Has the advantage of preserving incontinence
Treatment of choice for anal canal carcinoma	Chemoradiation

HIRSCHPRUNG'S DISEASE

MC association of Meconium plug syndrome	Hirschprung's disease
Acquired megacolon in children is commonly due to	Bad bowel habits
Failure of migration of neural crest cells in	Congenital megacolon
Hirschprung's disease is due to	Failure of migration of neural crest cells from cranial to caudal direction
True about hirschprung disease	Initially treated by colostomy
Hirschprung disease	Manometery excludes the disease
Hirschprung disease	Absent ganglionic cells in myenteric plexus of Auerbach, May involve small intestine rarely, Absent ganglionic cells in submucous plexus, Rectal biopsy is diagnostic
Aganglionic segment in Hirschprung disease at	Rectum
Aganglionic segment is encountered in which part of colon in case of Hirschprung disease	Distal to dilated segment
Hirschprung disease	Absent of ganglia in involved segment, Present only in infant and children
Congenital megacolon	Dilatation and hypertrophy of pelvic colon, Loud borborygmi, Symptoms appear in 3 days following birth, NO passage of stool
Constipation and abdominal distension for last 2 years in a child. Radiography reveals fecal matter containing distended bowel loops. a barium enema study shows a transition zone at rectosigmoid junction with reversal of rectosigmoid ratio	Hirschprung's disease
Failure to pass meconium within first 48 hours of birth in a newborn with no obvious external abnormality should lead to suspicion of	Congenital aganglionosis
NOT true about Hirschprung disease	Autosomal dominant
Most specific radiological feature of Hirschprung disease	Colonic dilatation
A male infant with distended abdomen shortly after birth passing of less meconium. Rectal biopsy is likely to show	Lack of ganglion cells
Differential diagnosis for Hirschprung disease	*Currarino triad (anorectal malformation, sacral base anomalies, presacral mass)*
Fluid used for rectal wash in Hirschprung disease	Normal saline
Operative treatment in hirschprung disease in only undertaken when child	Is at least 8kg in weight and thriving
Ideal age of surgery in long segment	*10 months to 1 year*

disease	
Treatment of choice for long segment disease	*Restorative proctocolectomy*
Duhamel operation	Hirschprung disease
Procedures for Hirschprung disease	Duhamel, Soave, Swenson
Surgery NOT done for Hirschprung disease	Bayar's disease

DIVERTICULOSIS AND DIVERTICULITIS

Massive bleeding per rectum in a 70 year old patient is due to	Diverticulosis
Massive bleeding from lower GI tract	Diverticulosis
Diverticulosis	Arise from diverticula of colon, Protrusion of mucosa through muscular layers at point of blood vessel
MC cause of diverticulosis of sigmoid colon	Bleeding PR
MC cause of colonic fistula in India at the age of 27 years	Crohn's disease
Commonest intestinal fistula with colonic diverticulum	Colovesical
Common cause of colovesical fistula would be	Crohn's disease
Commonest site for colonic diverticula	Sigmoid colon
Duodenal diverticulum is seen in	2nd part of duodenum
Treatment of bleeding duodenal diverticulum	*Diverticulectomy*
Small bowel diverticula	Seen on mesenteric border
Massive colonic bleeding in a patient of diverticulosis is from	Superior mesenteric artery
Diverticular disease NOT common in	Stomach
MC complication of diverticulosis of sigmoid colon	Bleeding per rectum
Diarrhea in enterocolic fistula due to	Decreased absorptive surface area
Colonic diverticulosis best diagnosed by	Barium enema
Sigmoidoscopic finding in acute diverticulosis of colon	Mucosa is inflamed
Diverticulitis	Left sided colon involvement is more common, Operation is NOT for all patients
Hinchey classification of Diverticulitis	**Type I – perforated paracolic abscess, type II – perforated distant abscess, type III – non communicating perforated diverticulitis with fecal peritonitis, type IV – perforating diverticulitis with free communication with peritonitis**
Management of type I and type II diverticulitis	**Resection and anastomosis**
Management of type III and type IV diverticulitis	**Haartman procedure**

ANGIODYSPLASIA

Massiva GI bleeding associated with aortic stenosis	*Angiodysplasia*
Angiodysplasia	Involvement of cecum, Age group > 40 years, Cause of troublesome lower GI hemorrhage
NOT true about angiodysplasia	Involvement of rectum in 50% of cases

Diagnosis of angiodysplasia by	*Intraoperative illumination of caecum*
Investigation of choice for angiodysplasia	*Mesenteric angiography*

MECKEL'S DIVERTICULUM

Meckel's diverticulum is derivative of	Proximal part of Vitellointestinal duct
Meckel's diverticulum arise from	Antimesentric border
Vestigial remnant of Vitellointestinal duct	Meckel's diverticulum, enterocyst, enteroteratoma of umbilicus
MC congenital anomaly of GIT in India	Meckel's diverticulum
Painless lower bleed in children	Meckel diverticulum
MC presentation of meckel diverticulum	Lower GI bleeding
A female complains of periumbilical pain and nausea particularly after taking food	Meckel's diverticulum
Meckel diverticulum is situated	60 cm from ileocaecal valve
Meckel diverticulum arise from	Ileum
Features of Meckel's diverticulum	Bleeding, Intussusceptions, Arises from antimesentric border, Located 60 cm from cecal valve, Perforation occurs, Occurs in 2% of population, Contains ectopic gastric tissue, Heterotopic gastric mucosa can ulcerate and cause a brisk gastrointestinal bleed, A fibrous band between the apex and umbilicus can cause intestinal obstruction, Has three layers with artery, Bleeding per rectum, 2 feet proximal to ileocecal valve on antimesenteric border, Can be a leading point of intussusceptions
Meckel's diverticulum	Patent Vitellointestinal duct, Lacks mesentry, Ectopic Pancreatic tissue
NOT true about meckel's diverticulum	Diarrhea very common, Contains of mucosa without muscle coat, Always gastric epithelium
Complications of meckel diverticulum	Hemorrhage, Intussusceptions, Strangulation
Diagnosed by Tc99 pertechnate scintigraphy	Meckel's diverticulum
Investigation of choice for Meckel's diverticulum	Tc99 Pertechnate scan
Best diagnosis for ectopic gastric mucosa of meckel's diverticulum	Radionuclide scan
A wide mouth meckel diverticulum found accidentally on laparoscopy	Leave it as such

INTESTINAL TUBERCULOSIS

GI is more common in ileocaecal region because	Alkaline pH, abundant lymph follicles,stasis of chime in ileum
Commonest site of tuberculosis in intestine	Ileum
Commonest site involved in ileocaecal TB	Intestinal mucosa
Ileocaecal tuberculosis	Rapid emptying of narrowed terminal ileum, Inverted umbrella sign, Stellate ulcers with elevated margin, Napkin ring stenosis
Intestinal tuberculosis	Causes intestinal perforation, Commonly associated

	with pulmonary TB, Caused by mycobacterium
Ileocaecal tuberculosis associated with	Megaloblastic anemia
Small intestinal tuberculosis can cause	Diarrhea, stricture, malabsorption
Hyperplastic ileocaecal tuberculosis	Mass in right iliac fossa, Common site in ileocaecal junction, X ray show indrawing of caecum from ileum, Conservative management is NOT done
MC cause of perforation of distal ileum in India	Tuberculosis
NOT true about hyperplastic tuberculosis	Surgery is the treatment of choice
Investigation for ileocaecal TB	*Barium meal follow through*
Barium follow through features of ileocaecal tuberculosis	Pulled up caecum, Widening of ileocaecal angle, Strictures involving terminal ileum
Pulled up Caecum	Ileocaecal Tuberculosis
NOT a diagnostic feature of ileocaecal tuberculosis on barium follow through	Apple core sign
MC indication of laparotomy in intestine TB	Intestinal obstruction
Contraindication for operating on Intestinal TB	Active disease

RECTAL ULCER

MC site of solitary rectal ulcer	*Anterior wall*
Feature of solitary rectal ulcer	*Lamina propria is infiltrated with collagen*
NOT true about solitary rectal ulcer	Lamina propria infiltration with lymphocyte, Involves posterior wall
NOT a treatment of solitary rectal ulcer	Banding, Sclerotherapy

RECTAL POLYP

Rectal polyp in adult associated with	Familial adenomatous polyposis coli
Rectal adenoma associated with	Hypokalemia
MC type of rectal polyp	Tubular adenoma
Rectal polyp commonly presents as	Bleeding
MC cause of fresh bleeding per rectum in 5 year child	Rectal polyp

RECTAL PROLAPSE

Muscle primarily responsible for rectal incontinence	Puborectalis
Resting tone of rectum decreased in	Micturition, Prolapsed rectum, Trauma involving perineum
Resting tone of rectum NOT decreased in	Retained feces in rectum
Rectal incontinence is due to involvement of	External anal sphincter, Internal anal sphincter, Pubococcygeus
MC site of solitary rectal prolapse	*Rectal polyp*
Surgery for rectal prolapse depends on	Sphincter function, degree of prolapsed
Treatment of rectal prolapse in childhood	Thiersch wiring
Recurrent rectal prolapse of rectum treated with	Ripstein operation

Delorome’s procedure for	Rectal prolapse
Young male patient with complete rectal prolapse	Abdominal rectopexy
Surgery for rectal prolapsed associated with least recurrence	Rectopexy
Lahaut procedure	*Rectal sling procedure*
Surgeries for rectal prolapse	*Ripstein, Wells, Lahaut*

ANATOMY OF ANAL CANAL

Felt anteriorly in per rectal examination in males	Bulb of penis, Prostate, Seminal vesicle when enlarged
NOT felt anteriorly in per rectal examination in males	Internal iliac lymph nodes
NOT felt in per rectal examination	Ureter
Anal canal	Supplied by pudendal nerve, Drained by veins forming portosystemic anastomosis, Part below dentate line is supplied by inferior rectal artery
Anal canal	Puborectalis is essential to maintain incontinence, Internal sphincter remains in state of tonic contraction
Anorectal angle is straightened during	**Straining**
Anal mucosal cushions	*3’O clock, 7’O clock, 11’O clock*
NOT true about anal canal	Completely lined by stratified squamous epithelium
Anal Triangle is NOT bounded by	Terminal part of Anal Canal
Dentate line	Glands of morgagni open below the line, Anal glands open at the line, Dentate line lies 2 cm above anal verge, Transitional epithelium lies BELOW dentate line
Pectinate line is between	Superior and inferior part of anal canal
Pectinate line divides	Nerve supply, Epithelium, Lymphatic and venous network
Line between upper mucous and middle transitional zone (pecten) of anal canal	Dentate line
Dentate line is fusion of	*Proctodeum and post allantoic gut*
Below pectineal line, lymphatic spread is	Superficial inguinal
Upper half of anal canal	Insensitive to pain
Lower limit of middle transitional zone (pecten)	White line of Hilton
Parts of external anal sphincter	*Deep, superficial, subcutaneous*
External anal sphincter is supplied by	S2,S3,S4 (pudendal nerve)
Internal anal sphincter is a part of	Internal circular fibres of rectum
Internal anal sphincter is supplied by	**Myenteric plexus**
Treatment of anal incontinence	*Gracilis neosphincter / dynamic graciloplasty*
Proctalgia fugax is possible due to	*Segmental cramps of pubococcygeus*
Anal column contains	*Superior rectal vessels*
Imperforate anus is found in a child. Child also have the risk of	*Tracheoesophageal fistula*

PILONIDAL SINUS

NOT a feature of pilonidal sinus	Recurrence uncommon

Treatment of pilonidal sinus	Surgical excision of sinus tract

HEMORRHOIDS

Superficial hemorrhoidal vein	No valve, Drain into inferior mesenteric vein
Hemorrhoids are	Venous dilatations
Prinicipal and earliest symptom of hemorrhoids	*Bleeding*
Hemorrhoids that prolapse and reduce spontaneously	*2nd degree*
FALSE about Piles	Always painful
NOT true about hemorrhoids	They are arteriolar dilatations
External hemorrhoids	Painful, below dentate line
External hemorrhoids below dentate line	Painful
Nerve mediating pain in external hemorrhoids	Pudendal nerve
Pain sensation in external piles mediated by	Pudendal nerve
Five day self subsiding pain is diagnostic of	Thrombosed external hemorrhoids
Internal hemorrhoids are arranged in 3, 7 and 11'O clock with respect to	*Superior rectal artery*
Best investigation to diagnose piles	Proctoscopy
Young office executive, bright red painless bleed since 7 days and abdominal pain. External and per rectal examination are normal. next step	Proctoscopy
Sitz bath consists of	Sits in a basin containing warm antiseptic solution
Treatment of primary piles	Surgery, Sclerotherpy
Solution for Hemorrhoids	5 % phenol in almond or arachis oil
Injection sclerotherapy ideal for	Internal hemorrhoids
Injection sclerosant used in 1st degree hemorrhoids through	*Gabriel syringe*
Barron banding for	*Second degree hemorrhoids*
Common complication after hemorrhoidectomy	Urinary retention

ANAL FISSURE

MC site of anal fissure	*Middle posterior*
Sentinel pile indicate	Anal fissure
Rectal examination should NOT be done in	Anal fissure
Anal fissure best diagnosed by	History and superficial clinical examination
Internal sphincterotomy is the treatment of choice for	Fissure in ano
Treatment of choice in Fissure in Ano	Fistulectomy
NOT a treatment of acute fissure in ano	External sphinterotomy

FISTULA IN ANO

Causes of persistence of sinus or fistulae	Foreign body, Non dependent drainage, Unrelieved obstruction, Presence of malignancy

Type of malignancy common in anorectal fistula	Squamous cell carcinoma, Adenocarcinoma
MC cause of fecal fistula	Previous surgery
Multiple fistula in ano commonly occurs in	Tuberculosis, Lymphogranuloma venerum
High or low fistula in ano is termed according to its internal opening present with reference to	Anorectal ring
MC type of fistula in ano	Intersphinteric
Goodsall's rule for	*Fistula in ano*
AIDS patient, CD4 less than 50, fistula in ano, treatment is	Seton
Treatment of choice for fistula in ano	Fistulotomy
Treatment of choice in Low sphinteric fistula near anal verge	Anal dilatation

ANORECTAL ANOMALIES

Cloacal anomaly	*High anomaly*
Anorectal anomalies associated with	Cardiac anomalies
X ray to detect congenital anorectal malformation at	24-48 hours
Invertogram taken after	6 hours after birth
A neonate is brought with history of not having passed meconium on examination there is no anal opening but a dimple. investigation of choice	Invertogram
A newborn girl not passed meconium for 48 hours has abdominal distension and vomiting. initial investigation of choice	Lower GI contrast study
A newborn baby not passed meconium for 48 hours since birth presents with vomiting and distension of abdomen. Most appropriate investigation	Barium enema (lower GI contrast study)
Treatment of absent anal orifice and meconuria	Transverse colostomy

ANORECTAL AND PERIANAL ABSCESS

Most common cause of anorectal abscess	Inflammation of anal gland
Common type of anorectal abscess	Perianal
Drain used for anorectal abscess	**Mallenkot drain**
Perianal abscess	Evacuated immediately
Ischiorectal fossa	Communicates with other side posteriorly
Increased incidence of infection in ischiorectal fossa	Poor blood supply
Staphylococcus in stool	*Ischiorectal abscess*
Nerve affected while incising ischiorectal abscess	Inferior rectal nerve

SUPERIOR MESENTERIC ARTERY SYNDROME

Superior mesenteric artery syndrome	Caused by compression of distended duodenum, Common in young females, Does NOT occur in obese individuals
In superior mesenteric artery embolism	Pain is out of proportion to physical findings

NOT true about superior mesenteric syndrome	Most common in 6th to 7th decade

PNEUMATOSIS INTESTINALIS

Gas in colonic wall	Pneumatosis coli, Necrotising enterocolitis, Hirschprung's disease
Gas filled cysts found in subserosa or submucous or small intestine or colon	Pneumatosis cystoides intestinalis
Pneumatosis cystoides	Spontaneous regression, May cause tension pneumoperitoneum, May cause severe bleeding, Surgery is NOT indicated
Surgery in pneumatosis intestinalis	Not required
Meteorism is associated with	*Renal trauma*
Meteorism (tympanite)	*Gaseous distension of abdomen occurs, renal colic may occur, radiographic appearance is dramatic, intestinal obstruction is a differential diagnosis*

INTESTINAL STRICTURES

Treatment of choice in multiple intestinal strictures of segment of jejunum	Stricturoplasty
Multiple strictures in intestine	Radiation enteritis

INTESTINAL MANIFESTATIONS OF TYPHOID

Typhoid ulcers	Bleeding perforation, Longitudinal orientation
14 year old girl with history of prolonged fever and abdominal discomfort is observed to have splenomegaly and leucopenia. In course of disease she developed acute abdominal event and dies. Autopsy finding of typhoid ulcer	Longitudinal ulcer
15 year girl, regular swimmer, sudden onset of abdominal pain, abdominal distension, fever of 39*C obliteration of liver dullness	Recurrent typhoid ulcer
Pyrexia for 10 days, develops acute pain in periumbilical region spreading all over the abdomen. most likely cause	Typhoid enteric perforation and peritonitis
15 year old girl, sudden onset of pain in abdomen, abdominal distension and fever of 39*C and obliteration of liver dullness	Ruptured typhoid ulcer
Typhoid perforation occur in	3rd week
NOT a complication of typhoid ulcer	Stricture, Multiple transverse ulcer
Typhoid perforation is diagnosed by	Plain X ray of abdomen in erect posture

COLONOSCOPY

Commonest site of perforation during colonoscopy	Sigmoid colon

ILEOSTOMY AND COLOSTOMY

Parastomal hernia is most frequently seen with	End colostomy
Most common serious complication of end colostomy	Parastomal hernia
NOT true about colostomy	Double barreled colostomy is commonly done nowadays
Early postoperative complication of ileostomy	Necrosis

INTESTINAL AMOEBIASIS

MC site of amoebic ulcer in intestine	Caecum
Flask shaped ulcer	Amoebic colitis
NOT true about amoebic ulcer	Perforation is common
Investigation of choice for invasive amoebiasis	ELISA
Treatment of amoebic colitis	Intravenous metronidazole

DIARRHOEA AND CONSTIPATION

IRRITABLE BOWEL SYNDROME

Young girl, abdominal pain and a recent change in bowel habit, passage of mucus in stool, NO blood in stool symptoms are increased with stress	Irritable bowel syndrome
Feature of irritable bowel syndrome	Abdominal distension, Diarrhoea, Constipation, Associated with anxiety, Stress predispose
NOT a feature of irritable bowel disease	Rectal bleeding
NOT true about irritable bowel syndrome	Predispose to malignancy in long term
Rome's criteria for	**Functional bowel disorders**
Best treatment for irritable bowel syndrome with spastic colon	Dietary fibre
Treatment of irritable bowel syndrome	**Rifamixin, Lubiprostone**
Treatment of Irritable bowel syndrome	Amitryptyline, Dicyclomine, Cisapride
Drug used for irritable bowel syndrome of constipation type	Lubiprostone

DIARRHEA

MC cause of death in children in age group 1-4 years	Diarrhea

Foods causing persistent diarrhea	*Apple juice, pear nectar*
Non inflammatory diarrhea	**Proximal small bowel, enterotoxin, ETEC, EAEC, cholera, clostridia, bacillus, S.aureus**
Inflammatory diarrhea	**Distal small bowel, cytotoxin, EIEC, EHEC, Shigella, Salmonella, Campylobacter, Yersinia**
Penetrating	**Salmonella, Yersinia**
Causative agent of Infant diarrhea	E.coli
Invasive diarrhoea	Aeromonas, Shigella, Rotavirus
Viral gastroenteritis is caused by	**Rotavirus, Norwalk virus, norvovirus**
Non invasive diarrhea	Bacillus cereus
Chronic diarrhea for minimum period of	*4 weeks*
Ruling out Chronic Diarrhea	Small Intestinal Biopsy
Osmotic diarrhea seen in	Lactase deficiency
Traveller's diarrhea	Diphenoxylate, Atropine
Drug used for Traveller's diarrhea	**Bismuth subsalicylate**
Effortless diarrhea is seen in	Cholera
Secretory diarrhoea associated with	Cholera, Laxatives, Excessive phenophthalein intake
Secretory diarrhea is caused by	Shigella infection
Secretory diarrhea	Stool volume>1L per day, normal osmotic anion gap, painless
Chronic secretory diarrhea	Carcinoid syndrome
Intractable diarrhea in children may be caused by	Cystic fibrosis, Secreting tumors, Milk allergy
Pseudodiarrhea	**Frequent passage of small volume of stool**
A two year old child presents with persistent diarrhea acidic stools and presence of one percent of reducing substance in fresh stools. Most probable diagnosis	Lactose intolerance
NOT a feature of lactose intolerance	*Hypoglycemia*
MC cause of seizures in children with diarrhoea	Hyponatremia
Convulsion in a child with dehydration and vomiting can only be due to	Decreased serum sodium
Diarrhea is a side effect of	Misoprostol
Antacid typically causing diarrhea	Magnesium hydroxide
NOT a cause of diarrhea in seropositive case of infection	Acanthamoeba
Indications for use of antibiotics in acute diarrhoea	Febrile dysentry with fever > 38.5, Immunocompromised patient, Elderly
Diarrhea responding to antibiotic therapy	Tropical diarrhea, Salmonella diarrhea
Drug of choice for chemotherapy induced diarrhea in AIDS	Octreotide
Atropine is added to Diphenoxylate to	Discourage abuse of preparation
MC cause of dysentery	Shigella dysenteriae
Bacillary dysentery is NOT associated with	Vomiting
Edema of rectal mucosa is reduced by	**Gauze impregnated with a warm solution of saturated magnesium sulphate**
Racecadotril	*Encephalinase inhibitor used in diarrhoea*
Diet for persistent diarrhea	*Diet A – reduced lactose, diet B – lactose free and reduced starch, diet C – monosaccharide based diet*

DEHYDRATION

Degree of dehydration	Mild – less than 5% of total body weight, moderate – 5 – 10% of total body weight, severe - >10% of total body weight associated with floppiness
Most likely organism causing some dehydration	Rota virus
Most dangerous dehydration	Hypernatremic
Hypernatremic dehydration	Serum Na > 150 mmol/L, Signs of dehydration are minimal
Features of hypernatremic dehydration	Irritability, Lethargy
Hypernatremic dehydration	Diuretic therapy, Colonic diarrhea, Excessive sweating
A month old formula fed infant has been brought with complains of watery diarrhea of 2 days duration and irritability of one day duration, receiving WHO ORS at home, markedly irritable child with a rather doughy skin and rapid pulse.	Hypernatremic dehydration
In a patient who has diarrhea and vomiting with inadequate water intake is suffering from	Extracellular dehydration with hyponatremia
NOT due to osmolar change in blood in dehydration	Hypotonia
Best parameter to monitor response to injection of a bolus dose of fluids in a dehydrated child	Radial Pulse
Most sensitive indicator of intravascular volume depletion in infant	Heart rate
NOT a reliable parameter of dehydration in a malnourished child	Skin turgor
2 year child 6.75 kg, skin pinch	Cannot be evaluated
6 month old child acute watery diarrhoea without signs of dehydration	Mothers milk, ORS
Advice for infant with mild diarrhea	Continue breast feeding
Irritable but drinking fluid	Oral rehydration therapy
A 5 year old boy passed 18 stools in last 24 hours and vomited twice in last 4 hours. He is irritable but drinking fluids. optimal therapy for this child	Oral rehydration therapy
A 5 year old boy passed 18 loose stools in last 24 hours and vomited twice in last 4 hours. irritable but drinking fluids.	ORS
Medical discovery of 20th century	ORS
Dehydration requiring ORT	Any dehydration
Amount of ORS to be given in 1st 4 hours in some dehydration	75 ml/kg body weight
A 3 month 4 kg, some dehydration. Amount of ORS given in first few hours	300 ml
Volume of fluid to replaced in a 10 kg child of obvious dehydration in first 4 hours	750 ml
Treatment of choice in severe dehydration	Ringer lactate
Treatment of hypernatremic dehydration	*Normal saline (to replace intravascular sodium)*

ORAL REHYDRATION SALT

Sugar salt solution	*Sucrose 40g and salt 4 g*
Super ORS	*Rice based ORS*
Oral rehydration mixture contains glucose and sodium because both of them	Facilitate absorption of each other from intestinal mucosa to blood
WHO ORS contains	Potassium chloride 1.5 g, Glucose 20 g, Sodium chloride 3.5 g, Sodium Potassium citrate 2.9 g
Glucose in WHO ORS	111
Sodium in WHO ORS	90
Total osmolarity of WHO ORS	311
Osmolality of WHO ORS	330
Value of chloride in ORS	80
Concentration of sodium in mmol/l in low osmolar ORS	75
Concentration of K+ in ORS	20
Content of citrate in ORS	10
Citrate in ORS	Increases shell life
Concentration of sodium chloride in ORS	3.5 gm
In ORS least amount is of	Potassium chloride
Sodium content of ReSomal (rehydration solution for malnourished child)	45 mmol/L
Freshly prepared ORS should NOT be used after	24 hours

CONSTIPATION

Intestinal hypomotility is seen in	Diabetes, Parkinsonism, Amyloidosis
Drugs causing constipation	Verapamil, MAO inhibitors, Tricyclic antidepressants, Ferrous sulphate
Causes constipation	Verapamil
Constipation is NOT caused by	Neostigmine
NOT a cause of chronic constipation in infant	High carbohydrate diet
Fecal soiling in children is commonly due to	Chronic constipation
Diet helping in relieving constipation	Non starch polysaccharides
Bisacodyl is converted to active form by	Endogenous esterases in bowel
Metabolism of bisacodyl	Hydrolysis of intestinal enzymes
Instant enema is given in	Habitual constipation
Prune juice is	**Laxative**

APPENDIX

GENERAL FEATURES OF APPENDIX

Appendix	Develops from midgut, Supplied by appendicular branch of ileocolic
Commonest anatomical position of appendix	Retrocaecal

Missed appendix	*Post ileal*
MC tumor of appendix	Carcinoid
Most commonly associated with Pseudomyxoma peritonei	Appendix

APPENDICITIS

Left sided appendicitis	Diverticulitis
Acute appendicitis is due to	Fecoliths, Worms of ileocaecal region
Earliest symptom of acute appendicitis	Pain
Most important sign in acute appendicitis	Pain in right lower abdomen
Usual order of occurrence of symptom in Acute appendicitis	Pain, Nausea, Local iliac tenderness
Appendicitis is associated with	*Moderate leucocytosis*
NOT true about appendicitis	Early antibiotics prevent rupture
Rovsing sign	Acute appendicitis
Rovsing sign	Pain in right lower quadrant during palpation of left upper quadrant
Aaron sign	Acute appendicitis
Ten horn syndrome	*Acute appendicitis*
When rectum is inflated with air through a rectal tube, pain and tenderness occur in right iliac fossa in case of appendicitis	Bastedo sign
Pain in acute appendicitis commonly referred to	Umbilicus
Attempts to straighten out a flexed thigh causes great pain in a patient with appendicitis. This is due to position of appendix near	*Psoas muscle*
In case of retrocaecal appendicitis movement aggravating pain	Extension
Frequent mechanism in perforation of appendix	Tension gangrene due to accumulation secretions
Diffuse peritonitis following appendicitis is usually seen	When appendicular perforation occurs early within 24 hours
Worst prognosis of appendicitis in elderly and children because	*Omentum and peritoneal cavity appear to be less efficient in localizing disease in each groups*
Alvarado scale is used in diagnosis of	*Appendicitis*
Pathological feature of acute appendicitis	*Neutrophils within muscular wall*
Appendicitis diagnosed by	TLC, DLC, USG, CT
USG sign of acute appendicitis	Non compressible
Visual appendix in USG is suggestive of	Acute appendicitis
NOT useful in appendicitis	Purgation
A patient with Crohn's diseases was opened for and an inflamed appendix is found. treatment of choice	Appendicectomy
McBurney incision	Most suitable if the diagnosis of appendicitis is definite, It is converted to muscle cutting incision it is called Rutherford Morrison incision, Inguinal hernia may be a sequale of incision

Nerve commonly injured during Mcburney's incision	Iliohypogastric
NOT true about Mcburney incision	Incision can be extended upwards and downwards
Muscle splitting incision	Lanz
A gridiron incision becomes Rutherford Morrison's incision if extended by	Cutting muscles laterally
Appendicular stump obtained by	Ligation and invagination
If base of appendix is inflamed	No burying of stump
Laparoscopic appendix removal technique	*Endoloop technique*
NOT an Early complication of appendicectomy	Sterility
An appendicular fistula is least likely to heal if	There is stenosis or narrowing of sigmoid colon

TUMOURS OF APPENDIX

Generalized abdominal pain, fever, recurrent vomiting, temperature 103*F, right ililac fossa tenderness, WBC 19600/mm3	Ruptured appendicular abscess
Oschner Sherren regimen is used in management of	Appendicular mass
MC neoplasm of appendix	Adenocarcinoma
25 year old patient with mass in right iliac fossa. laparotomy shows carcinoid of 2.5 cm diameter	Right hemicolectomy

INFLAMMATORY BOWEL DISEASE

GENERAL FEATURES OF INFLAMMATORY BOWEL DISEASE

Atypical colitis	**Collagenous colitis, lymphocytic colitis**
Typhilitis	**Neutropenic enterocolitis**
Inflammatory bowel disease	Seronegative arthritis
Treatment of Inflammatory Bowel Disease	5-aminosalicylic acid derivatives
Sulfa drug used in inflammatory bowel disease	Sulfasalazine
Recurrent episodes of bloody diarrhea for 5 years, despite regular treatment with sulphasalazine, several exacerbations of his disease. Required several weeks of steroids for control of flares	Azathioprine
Drug NOT give in Inflammatory bowel Disease	Cotrimoxazole
Treatment of complicated regional ileitis	Steroids, non absorbable sulfonamides, radiotherapy

CROHN'S DISEASE

Mutation in NOD2 gene is associated with	*Crohn's disease*
Crohn's disease may be caused by	Clostridium difficle
Crohn's disease also known as	Regional Ileitis
Crohn's disease is associated with	Non Caseating granuloma
Child abdominal pain, fever, bloody diarrhea, stool culture negative for enteropathogens, sigmoidoscopy normal. Child has an episode of renal colic and passed	Crohn's disease

urinary gravel. Mantoux test 5*5 mm	
14 year old girl with history of abdominal pain, postprandial, passing blood in stool, fever, weight loss since ten months. episodes of passing blood in stools	Crohn's disease
Diarrhoea and blood in stool presents with multiple fistula in the perineum and multiple stricture in small intestine	Crohn's disease
Crohn's disease	Through and through involvement of bowel wall
Transmural involvement & Granuloma	Crohn's disease
Transmural inflammation with skip lesions	Crohn's disease
Skip lesion with tuberculoid granuloma	Crohn's disease
Colovesical fistula commonly arise from	Crohn's disease
Colonic obstruction is frequent in	**Crohn's disease (segmental asymmetric colitis with stricture) - irony**
Lead pipe appearance of colon on barium enema	Crohn's involvement of colon
Bull's Eye Lesion, Target Lesion, String Sign of Kantor, Comb Sign (Prominent Vasa Recta)	Crohn's disease
String of beads appearance	Crohn's disease
Cobblestone appearance	Crohn's disease
Lead pipe appearance	Ulcerative colitis
Creeping fat	**Crohn's diseae**
Fistula is common in	Crohn's disease
Anal lesions are first manifestation of Crohn's disease in	25%
NOT a feature of Crohn's disease	Lymphoid hyperplasia, Continuous mucosal damage
NOT seen in Crohn's disease	Hyperplastic polyp, Diverticulosis
MC cause of death in Crohn's disease	Malignancy
Extraintestinal manifestation of Crohn's disease	Skin nodules, Uveitis, Sclerosing cholangitis
MC renal Complication of Crohn's disease	Nephrolithiasis
Antisaccharomyces cervesiae antibody	Crohn's disease
Established biological therapy for crohn's disease	Anti TNF alpha antibody
Useful in treating Crohn's disease	Infliximab
A patient of Crohn's disease, underwent resection anastomosis. Now presents on 7th post-op day with anastomotic site leak from a fistula. Everyday leakage volume adds up to 150-200ml. There is no intra-abdominal collection and the patient is stable without any complains. What will be the next line of management?	Do conservative treatment and leave him and hope for the spontaneous resolution

ULCERATIVE COLITIS

Pseudomalignant condition of Colonic carcinoma	Ulcerative colitis
Disease more common in non smokers	Ulcerative colitis
Smoking in ulcerative colitis	Protective
Appendicectomy is protective in	**Ulcerative colitis**
Ulcerative colitis involve	Mucosa and submucosa
Ulcerative colitis	*Diffuse mucosal inflammation*
Always involved in ulcerative colitis	Rectosigmoid
Ulcerative colitis starts in	Rectum
Chronic diarrhoea, cryptitis and crypt abscess	Ulcerative colitis

Ulcerative colitis associated with primary sclerosing cholangitis	May develop biliary cirrhosis, raised alkaline phosphatase, increased risk of hilar cholangiocarcinoma, PSC reverts after total colectomy
Primary Sclerosing Cholangitis is associated with	Ulcerative Colitis
Lead Pipe Appearance, Pseudopolyp, Backwash Ileitis	Ulcerative Colitis
Pipe stem appearance of Colon	Ulcerative colitis
Distension of colon in ulcerative colitis is associated with	*Loss of haustrations*
Stem pipe appearance in barium enema	Ulcerative colitis
Toxic megacolon, Lead pipe colon	Ulcerative colitis
Ulcerative colitis is associated with	Cryptitis
Toxic megacolon is seen in	Chronic non specific ulcerative colitis
Pseudopolyp	No risk
Collar button ulcer	**Ulcerative colitis**
Extraintestinal manifestation of Ulcerative colitis that does NOT respond to Colostomy	Primary Sclerosing Cholangitis
Risk of malignancy in ulcerative colitis is more if	Onset is in childhood, Extensive involvement of colon
In ulcerative colitis, carcinoma arise from	Dysplastic sites
Best morphological feature to distinguish between ulcerative colitis and crohn's disease	Diffuse distribution of pseudopolyp
Malignancy in ulcerative colitis is related to	*Duration of ulcerative colitis*
NOT seen in Ulcerative Colitis	Skip lesions
Epitheloid like giant cells NOT seen in	Ulcerative colitis
NOT associated with ulcerative colitis	Pancreatitis
NOT true about malignancy in ulcerative colitis	Left side is more common
NOT true about ulcerative colitis	Circinate balanitis
Not true about Ulcerative Colitis	Granuloma
NOT a complication of ulcerative colitis	Peptic ulceration
NOT a known complication of ulcerative colitis	Stricture
Barium enema finding in Chronic ulcerative colitis	Loss of haustral marking and ribbon pattern,Slip areas in descending colon and caecum,Rigidity and contraction of Caecal walls
Best treatment for remission of acute ulcerative colitis	Prednisolone
Sulfonamide used in treatment of ulcerative colitis	Sulfasalazine
Treatment of Ulcerative colitis	Salzopyrine/5 amino salicylic acid
Drug of choice for ulcerative colitis	Sulfasalazine
Sulfasalazine acts on	**Colon**
Mesalazine acts on	**Distal ileum and colon**
Mesalazine is used in treatment of	Ulcerative colitis
Ulcerative colitis patient remission after treatment with sulphasalazine. He requires steroid. next line of treatment	Azathioprine
Surgery in ulcerative colitis	Done in cases where medical treatment fails, Pouch surgery done
Treatment of Choice for Ulcerative Colitis (Elective)	Restorative Proctocolectomy with Ileal Anal Pouch Anastomosis
Treatment of choice in chronic ulcerative colitis	Proctocolectomy with ileoanal anastomosis
Treatment of choice in ulcerative colitis with acute perforation	Total colectomy and ileostomy
Commonest Complication following ileoanal	Pouchitis

anastamosis for ulcerative colitis	
NOT a surgical indication for inflammatory bowel disease	Extraintestinal complication
Surgery associated with lowest rate of recurrence in ulcerative colitis	*Complete proctocolectomy with Brook's ileostomy*
Pyoderma Gangrenosum most commonly associated with	Ulcerative colitis
Cribriform healing of lesion	*Pyoderma gangrenosum*
Treatment of pyoderma gangrenosum	Surgery and antibiotics

INTESTINAL OBSTRUCTION

GENERAL FEATURES OF INTESTINAL OBSTRUCTION

Commonest cause of acute intestinal obstruction	Adhesions
MC cause of bowel obstruction	Peritoneal adhesion
Most suggestive of neonatal small bowel obstruction	Bilious vomiting
MC cause of small intestinal obstruction	Iatrogenic adhesions
Most common cause of acute intestinal obstruction in neonate	Duodenal atresia
Commonest cause of intestinal obstruction in children	Intussusceptions
Commonest cause of intestinal obstruction in newborn	Duodenal obstruction
Commonest cause of intestinal obstruction in 30 year old female	Post op adhesions
Causes of obstruction in large bowel	Diverticulitis, carcinoma, volvulus
Commonest cause of colonic obstruction	Neoplasm
Commonest cause of colonic obstruction in neonate	Aganglionic colon
Most suggestive of neonatal small bowel obstruction	Bilious vomiting
Number of fluid levels to state as abnormal	*3*
Adhesive intestinal obstruction	Avoid surgery for initial 48-72 hours
Acute intestinal obstruction	Vomiting is common in duodenal obstruction, In colonic obstruction distension is common than vomiting, X ray erect posture is diagnostic
First to appear in a case of intestinal obstruction	Colicky pain
Pain of intestinal obstruction is best described as	Cramping
Water loss is severe if intestinal obstruction occurs at	First part of duodenum
Distended abdomen in intestinal obstruction is mainly due to	Swallowed air
Rupture of hollow viscus	Absent peristaltic activity, free air in peritoneal cavity, abdominal pain, shoulder pain, nausea, vomiting
Clinical features of intestinal obstruction	Abdominal distension, Vomiting
Newborn presenting with intestinal obstruction, constipation, multiple air fluid levels	Duodenal atresia, ileal atresia, Ladd's band
Primary feature of small intestinal obstruction	High peristalsis with colic
A woman of 35 years old comes to emergency department with symptoms of pain in abdomen and bilious vomiting but no distension of bowel. No air fluid	Duodenal obstruction

level	
Patient with spinal POP cast presenting with bilious vomiting	Duodenal obstruction
Windsock web	*Duodenal obstruction*
In terminal ileal obstruction	Caecum is collapsed
Intestinal obstruction surgery and biopsy of ileal loop was done and sent for histology-it will contain	Villous adenoma
NOT a cause of duodenal obstruction syndrome	Malnutrition
Features of colonic obstruction	No passage of gas absolutely, Distension of abdomen, No passage of stools absolutely
In obstruction of large gut rupture occurs at	Cecum
Anismus	**Outlet obstruction due to non relaxing puborectalis**
NOT radiological diagnosis by USG in surgical obstruction	Peritoneal seeding
Features of small bowel obstruction	*Loops distended, 3-5 cm, loops in central position, many loops, short width, valvulae, no gas in large bowel loops*
Radiological feature of ileal obstruction	Microcolon on barium enema, Obstruction in barium meal
Best way to diagnose lower small intestinal obstruction	Multiple air gas shadows on X ray
Investigations needed in intestinal obstruction	Barium meal, Erect X ray abdomen, Supine X ray abdomen
Investigation of choice for suspected intestinal obstruction	X ray abdomen erect posture
Acute mechanical large bowel obstruction should be operated because of	Early gangrene and perforation, Closed loop obstruction and cecal perforation
Barium should never be given by mouth to a patient with a possible	**Colonic obstruction**
For intestinal obstruction, immediate operation should NOT be done in case of	Post op adhesion

INTESTINAL ATRESIA

Commonest site of intestinal atresia	Duodenum
Gastric aspiration of more than 20 ml in a child at birth suggests	Duodenal atresia
Congenital anomaly associated with duodenal atresia	Annular pancreas, Biliary atresia, Cardiac anomalies
Always present with bilious vomiting	Atresia of 3rd part of duodenum
Apple peel deformity, Christmas tree deformity	*Type IIIB type of jejunoileal atresia*
Characteristic X ray finding of Ileal atresia	Dilated loop of intestine with multiple fluid levels
Treatment of duodenal atresia	Duodenoduodenostomy

MALROTATION

Reduction of physiological hernia	10th week
Total rotation of midgut	*270* counter clock wise*
Caecum is found to be placed below stomach and in	Malrotation

midline. Abnormality must have taken place while rotation of gut	
A child has recurrent abdominal pain and bilious vomiting. Condition was diagnosed by barium follow through. Surgical procedure – ladd's band division, appendectomy and mesenteric widening	Malrotation
Malrotation presents as	Bleeding per rectum, Bilious vomiting
Symptoms of malrotation	*Volvolus, chronic abdominal pain, failure to thrive*
In incomplete rotation, rotation is arrest at	*180**
Ladd's band extends from	*Right upper quadrant to cecum*
Rare form of intestinal malrotation	*Hyper rotation*

PARALYTIC ILEUS

Causes of paralytic ileus	Post operative, Uremia, Peritonitis
MC cause of paralytic ileus	Postoperative
Paralytic ileus caused by	Peritonitis
Spastic ileus seen in	Porphyria
Paralytic ileus is seen in	Spinal cord injury, Uremia
Does NOT produce paralytic ileus	Morphine, Pethidine
Paralytic ileus causes	Hypokalemia, Abdominal distension, Multiple air fluid levels
Does NOT require immediate operative therapy	Paralytic ileus
NOT a feature of paralytic ileus	Loops of intestine NOT seen because of loss of peristalsis
Persistent paralytic ileus is treated by	Surgical reopening
Prolonged post op ileus is best treated by	Laparotomy and exploration

MECONIUM ILEUS

Soap bubble appearance	Meconium ileus
Snow storm ascites	Meconium ileus
Ground glass appearance in right lower quadrant	*Meconium ileus*
Meconium ileus associated with	Fibrocystic disease of pancreas
Disorder to be suspected when a newborn baby has meconium ileus	Cystic fibrosis
Fluid levels are NOT seen in	Meconium ileus
Gastrogafin enema has therapeutic advantage in	*Meconium ileus*

INTUSUSSCEPTION

MC cause of intestinal obstruction in 3	*Intususception*

months to 6 years	
Commonest cause of intussusception	Hypertrophy of submucous peyer's patches
Type of lipoma leading to intusussception	Submucous
Cause of bloody vomitus in neonate	Intusussception
Henoch Schonlein purpura may rarely cause	Intussusceptions
MC type of intussusception	Ileocolic
Least common type of intussusceptions	Multiple
Intusssuscepiens is	Outer layer
A 10 month old infant presents with acute intestinal obstruction. Contrast enema X ray shows intussusceptions. Likely cause	Peyer's patch hypertrophy
Previously healthy infant, recurrent episode of abdominal pain, passing altered stool after episode of pain. no history of vomiting or bleeding per rectum	Intusussception
Recurrent abdominal pain with intestinal obstruction and mass passes per rectum in favor of	Intussusception
A patient has acute abdominal pain with blood and mucus in stool with palpable mass per abdomen is due to	Intussusception
A 6 month old baby with history of bloody diarrhea of 2 days duration with abdominal distension and on examination the baby screams, diagnosis is	Intusussception
Infant, bloody stool, mass in abdomen	Intusussception
Red currant jelly stool	Intussusception
First and most common symptom of intusussception	Pain
Features of intussusceptions	Target sign, Coiled spring sign, Dance sign
Claw sign	Intusussception
Coiled spring sign	Intusussception
Signe de dance	Empty right iliac fossa in intussusceptions
Doughnut sign	Intusussception
Pseudokidney sign	*Intusussception*
Best investigation for ileocolic intusussception	Barium enema
Investigation of choice for	Intusussception
Intussusception	Relieved by barium enema
Complete reduction of intussusceptions is indicated by	Free passage of barium in terminal ileum

VOLVOLUS

Predisposing factors for sigmoid volvolus	Band of adhesion, Long pelvic mesocolon, Narrow attachment of pelvic mesocolon, Loaded pelvic colon
Volvolus	More common in psychiatric patient, Sigmoid volvolus is more common than caecal volvolus
Coffee bean sign	Volvolus
Borchardt triad with acute epigastric pain, violent retching and inability to pass nasogastric tube	Acute gastric volvulus
Apex of volvolus of caecum in plain X ray abdomen	Left iliac fossa
Volvolus occur more frequently in	Sigmoid colon
Bird of prey sign	Sigmoid volvolus
Bent inner tube appearance	**Sigmoid volvolus**
Rotation of sigmoid volvulus	Anticlockwise

Feature of Sigmoid volvolus	Vomiting
Distended ileal loops in a distended sigmoid volvolus	*Compound volvolus*
Bleeding per rectum NOT seen in	Sigmoid volvolus
NOT true about volvolus	Lower GI scopy is contraindicated in sigmoid volvolus
Best investigation for volvolus neonatarum with suspected perforation	Plain X ray
Definite treatment of sigmoid volvolus	Colectomy
Paul Mickulicz procedure for	*Volvolus*
Bishop Coop's operation	*Ileoileostomy*
Surgery done in caecal volvolus if cecum is ischemic	*Right hemicolectomy*
Treatment of cecal volvolus	*Operative detorsion, right hemicolectomy with ileocolostomy*

INTESTINAL PSEUDO OBSTRUCTION

Colonic pseudo obstruction	Hypothyroidism, Diabetes mellitus, Dermatomyositis, Scleroderma
A 56 year old woman not passed stools for last 14 days. X ray shows no air fluid level. probable diagnosis	Intestinal pseudo obstruction
Acute pseudo obstruction of colon is also known as	Ogilvie syndrome
NOT true about Ogilvie syndrome	It is caused by mechanical obstruction of colon
Colonic pseudo obstruction does NOT occur in	Hyperthyroidism

DIAGNOSIS OF INTESTINAL OBSTRUCTION

Single bubble appearance	Pyloric stenosis
Double bubble sign	Annular pancreas, Ladd's band, Duodenal atresia, Pancreatic pseudocyst
Triple bubble sign	Jejunal obstruction
A newborn presenting with intestinal obstruction showed on abdominal X ray, multiple air fluid levels. NOT diagnosis	Pyloric obstruction

MALABSORPTION SYNDROMES

GENERAL FEATURES OF MALABSORPTION

Rumination disorder	*Chronic motility disorder, failure to thrive, regurgitation of food, no nausea, usually between 3 to 14 months of age*
Oast house disease	Methionine Malabsorption
Malabsorption is caused by	Giardia lamblia
Malabsorption syndrome caused by	Giardiasis, Stronglyoides, Capillaria phillipensis

NOT a malabsorption disease	Tangier's disease
Non absorption of fat soluble vitamins is due to	Steatorrhoea
Steatorrhea seen in all except	Obstructive jaundice
Malabsorption, iron deficiency anemia, complete villous atrophy	Antiendomyosidal antibodies
Chronic diarrhoea, anemia, raised liver enzymes	Anti endomysial antibody
Fat in stool > 6 gm/day indicate	Malabsorption
Carotenoids are ABNORMAL in	Malabsorption
Intestinal malabsorption is NOT produced	Intestinal tuberculosis
Screening test for fat malabsorption	Sudan III
Oral Glycine Glycolate test shows	Bile Malabsorption
Gold Standard test for Fat malabsorption	Quantitative estimation of Fecal Fat
Normal daily fecal fat excretion	*5 gram*
Best method of diagnosis of malabsorption disorder	Small intestinal biopsy
X ray finding of small intestinal malabsorption	Dilatation of bowel, Mucosal atrophy, Flocculation of barium
Biopsy features of Whipple and celiac disease	**Whipple disease – diffuse and specific, Celiac disease – diffuse and non specific**
NOT a barium study findings in malabsorption syndrome	*Stricture of bowel*
Increased stool osmotic gap	*Carbohydrate malabsorption*

D-XYLOSE TEST

D-XYLOSE TEST	D-Xylose is a carbohydrate completely absorbed in Small bowel without help of Pancreatic enzymes
Best test for small intestine absorption of carbohydrate	D xylose test
Altered Intestinal Mucosa (Non invasive)	Urinary D-Xylose Test
Positive D Xylose test	Malabsoption due to Intestinal mucosal disease
Positive D- Xylose test	Celiac sprue
Negative D Xylose test	Malabsorption due to Pancreatic disease
False positive xylose test	Bacterial overgrowth, Blind loop syndrome, Renal failure, ascites, Pyloric stenosis, Antibiotic therapy

CELIAC DISEASE

Celiac disease	Intestinal biopsy, Unequivocal response to gluten restriction
Celiac disease associated with	Dermatitis herpetiformis, Type I DM, Lymphoma, Atrophic gastritis
MC symptom of celiac disease in children	*Failure to thrive*
MC sign of celiac disease in children	*Wasted muscle*
MC extraintestinal manifestation of celiac disease	*Iron deficiency anemia*
Mildly increased incidence of NHL and 10-15 times increased incidence of squamous esophageal cancer	*Celiac sprue*

Celiac sprue is associated with	*Dermatitis herpetiformis*
MC cause of death in celiac disease	*Non Hodgkin's lymphoma*
NOT a feature of celiac disease	Increase in thickness of mucosa, Mucosal atrophy, Presence of giardia lamblia
NOT true about celiac disease	Increased brush border enzymes
Hypercalcemia NOT associated with	Celiac disease
Avoided in gluten associated enteropathy	Wheat, barley, rye, oat
Used safely in celiac disease	Rice, corn
NOT avoided in patients with celiac disease	Maize
Vitamin deficiency uncommon in celiac disease	Vitamin B12
Gluten sensitive enteropathy is strongly associated with	HLA-DQ2
Sensitive and specific for celiac disease	Anti endomyosial antibody
Anti transglutaminase is associated with	Celiac disease
Antigliadin antibodies	Celiac disease
Histologic features of celiac disease	Crypt hyperplasia, increase in intraepithelial lymphocytes, increase in inflammatory cells in lamina propria
Treatment of celiac disease	Gluten free diet
Marker used to monitor compliance with gluten free diet	*IgA*

WHIPPLE DISEASE

Whipple's disease	Macrophages with PAS positive material inside the lamina propria
Hallmark of Whipple disease	PAS positive macrophages
Whipple's disease	Increase in number of macrophages in mucosa, dilatation of lymphatics in marrow, lipid deposition in mucosa
Macrophage containing large quantities of undigested and partial digested bacteria in intestine	Whipple's disease
MC CNS manifestation of Whipple's disease	Dementia
NOT true about histological feature of whipple disease	Marked increase in number of intraepithelial lymphocytes
Organ NOT involved in Whipple's disease	Lung
Jejunal biopsy is used in	Whipple disease

SHORT BOWEL SYNDROME

In massive small bowel resection, intestine compensate by	*Lengthened individual villi*
Intestinal failure occurs in	*Short bowel syndrome*
Short bowel syndrome	Diarrhoea, weight loss, steatorrhoea
Short bowel syndrome is NOT characterized by	Hypogastrinemia
NOT a component of Bariatric surgery	Ileal resection
Complications of ileal resection	Megaloblastic anemia, Gastric hypersecretion, Malabsorption syndrome
Vitamin deficiency most commonly seen in short bowel	Vitamin B12

syndrome with ileal resection	
Renal calculus is seen in massive bowel resection due to	More oxalate absorption in gut
Resection of 90% of ileum and jejunum does NOT cause	Hypogastrinemia
NOT true about blind loop syndrome	*Surgery is always required*

TROPICAL SPRUE

Tropical sprue is caused by some strains of	E.coli
Vitamin deficiency found in tropical sprue	Folic acid
Intestinal biopsy is NOT diagnostic in	Tropical sprue

PROTEIN LOSING ENTEROPATHY

Protein losing enteropathy is seen in	Sprue, Ulcerative colitis, Whipple's disease
Protein losing enteropathy is diagnosed by	Tc albumin, Tc dextran
Confirmatory test for protein malabsorption	*Stool level of alpha 1 anti trypsin*

LIVER

ANATOMY AND PHYSIOLOGY OF LIVER

Weight of Human Liver	1400 – 1600 gm
Bile flows from	**Zone 3 to zone 1**
Daily basal secretion of hepatic bile	**500 – 600 ml**
Bare area of Liver is related to	Right Suprarenal gland
Part placed behind porta hepatis and in front of IVC and forms the roof of epiploic foramen	Caudate Process of Liver
Portal triad	*Bile duct, portal vein, hepatic artery*
Not a tributary of Portal Vein	Hepatic Vein
Structure crossing through calot's triangle	*Cystic artery*
Calot's Triangle is NOT bounded by	Hepatic artery
Liver	Functional unit is ACINI, Portal space contains bile duct, portal vein and hepatic artery. Zone 1 is well oxygenated, Central vein drains lobule
Stellate cells of Von Kupffer are seen in sinusoids of	Liver
Function of hepatic stellate cells	Vitamin A storage
Space of Disse in	Liver
Hepatorenal pouch of Morrison	*Right posterior intraperitoneal recess*
Dual blood supply	Liver
Maximum blood supply to liver by	Portal vein
Blood supply to liver	80% portal vein,20% hepatic artery
Percentage of blood flow to liver is supplied by hepatic artery	20%
Blood Flow of Liver in ml per minute	1500 ml

Aberrant right hepatic artery arise from	*Superior mesenteric artery*
Aberrant left hepatic artery arise from	*Left gastric artery*
Bilirubin is absent in urine because of	Lipophilic
Bilirubin is absent because	Non filtered and lipophilic
Conjugation of bilirubin to glucoronic acid in liver	Converts a hydrophobic compound into hydrophilic one, Able to cross cell membrane
Detergent action of bile salts is due to	Amphipathic
Predominantly mitochondrial liver enzyme	SGOT AST
Liver does NOT synthesize	Gamma globulin

LOBES OF LIVER

Fissure of gans	*Right lobe of liver*
Left hepatic artery supplies	*Left, quadrate and caudate lobe*
Corinaud nomenclature is based on	Hepatic vein and portal vein
Liver lobes are surgically divided by	Hepatic and portal vein
Line of surgical division of lobes of liver	Gall bladder bed to IVC
Liver is divided into two halves by	Portal vein, Hepatic artery, Common bile duct
Does NOT divide liver into two halves	Right hepatic vein
NOT a basis of function division of liver	Hepatic vein
Classical lobule of Liver is centered around	Central Vein
Caudate lobe of liver corresponds to which segment of Corinaud classification	Segment I (medial to IVC, drains into IVC)
Segment having independent vascularisation	Segment I caudate (IVC)
Relation of caudate lobe of liver	Anterior to right inferior phrenic artery
A surgeon excises a portion of liver to left of attachment of falciform ligament	Segment 2 and 3
Portion of liver left to falciform ligament	2,3
Segment 4 of liver in	Left lobe
Segment IV	Quadrate (medial to gall bladder, drains into gall bladder)
Segment IV of liver	Quadrate
Quadrate lobe is between	*Groove for ligamentum teres and gall bladder*
Right lobe of liver contains	5,6,7,8 segments
Segment 9 of liver according to corinaud classification	*Right paracaval portion of posterior liver*
Hepatic duct	Left hepatic duct formed in umbilical fissure, right hepatic duct formed from V and VIII segments. left hepatic duct crosses IV segment
Left hepatic duct drains	II,III,IV
Right hepatic ducts drain	5,6,7,8
Right hepatic duct does NOT drain	Segment III
Longest extrahepatic course	*Left hepatic duct*

GENERAL FEATURES OF LIVER

Source of hepatic stem cells	Oval cells
Alagille syndrome is associated with	*Jagged 1*
Pigmentation in liver	Lipofuscin, bile pigment, malarial pigment, pseudomelanin,Wilson's disease
Nutmeg liver	Chronic venous congestion
Honeycomb liver	Actinomycosis
Venoocclusive disease is associated with	*Pyrrotizide alkaloids (Crotalaria)*
Venoocclusive disease in hepatic vein is characterized by	Hepatomegaly, Hepatic fibrosis
Hepatomegaly is NOT a feature of	Hepatic porphyria
NOT a cause of hepatomegaly with liver tenderness and elevated diaphragm	Acute cholecystitis
Central necrosis of liver	Congestive cardiac failure, RHF
Toxic injury of liver affects	*Zone 1*
Mallory bodies is composed of	Intermediate filaments
Mallory bodies is NOT seen in	*Hemochromatosis*
Mallory hyaline is found in	Primary Biliary cirrhosis, Alcoholic hepatitis, Wilson's disease, hepatocellular carcinoma, Indian childhood cirrhosis
Mallory hyaline is NOT seen in	Secondary biliary cirrhosis
Overlap syndrome	**Primary biliary cirrhosis with autoimmune hepatitis**
Microvesicular steatosis is associated with	*Reye syndrome, acute fatty liver of pregnancy, hepatic encephalopathy, hepatic encephalitis, valproate, tetracycline*
Macrovesicular steatosis	**Insulin resistance, type II diabetes mellitus, glucocorticoids estrogen, tamoxifen, amiodarone, nutritional, Wilson's disease, chronic hepatitis C genotype 3**
Impaired hepatic blood flow in venous occlusive disease is due to	*Fibrous obliteration of terminal hepatic veins*
MC nodule found in liver	Hemangioma
MC benign tumour of liver	Hemangioma
Commonest incidentaloma in liver	Hemangioma
Hemangioma	*Mostly asymptomatic, centripetal fill in appearance is seen on post contrast study, angiography shows characteristic fill in appearance*
Investigation of choice for hemangioma	*Biphasic CT*
Investigation for hemangioma	*MRI*
Spontaneous rupture of liver occurs	Hepatoma
Primary treatment of choice for hepatoma in children	Surgery
MC cause of hepatic metastasis in children	Neuroblastoma
Multiple liver secondaries are common in	Gall bladder carcinoma, Obese
Hepatic avulsion injury	*Stage VI*
Scales for fibrosis of liver	**Metavir scale, Ishak scale**
Liver malignancies are studied by	Radionuclear study, Ultrasound, CT, Laparoscope

22 year old man solitary 2 cm space occupying lesion of mixed echogenecity in right lobe of liver on USG, next test	Hepatic scintigraphy
Solitary hypoechoic lesion of liver without septate or debris is most likely to be	Simple cyst
Contrast used in Liver scan	Rose Bengal
Radioactive isotope used in Liver scan	Technitium 99
USG shows focal lesion in liver. Next line of investigation	CT scan
Gold standard investigation for imaging liver	Spiral CT
Best modality to diagnose liver mass	USG
Wedge hepatic venous pressure represents pressure in	Sinusoids
Vascular inflow occlusion of liver	Pringle manoeuvre
Pringle maneuver is digital compression of	*Hepatic artery, portal vein and CBD (porta hepatis)*
Liver biopsy is done through 8th midaxillary line to avoid injury to	Lung
Contraindications for major hepatic resection for metastatic disease	Total hepatic involvement, Advanced cirrhosis, Extrahepatic tumor involvement
Minimum amount of normal perfused liver parenchyma to be left intact when a hepatic resection is planned	20%
Following resection of 2/3rd of liver, regeneration is complete with	4-6 months

LIVER ENZYMES

5' nucleotidase increased in	Cholestasis
Abnormal excretory functions of hepatocytes may be assessed by	Increased alkaline phosphatase, Increased gamma glutamyl transferase
Gamma glutamyl transferase is a marker of	Alcoholism
Increased serum alkaline phosphate	Obstructive jaundice, Paget's disease of bone, hyperparathyroidism, metastatic tumours in bone
Liver does NOT synthesize	Immunoglobulin
Abnormalities in synthetic function of liver	Increased prothrombin time, Hyperbilirubinemia, Acute phase reactants
Child presenting with obstructive jaundice	Gamma glutamyl transpeptidase, Alkaline phosphatase, 5' nucleosidase

UNCONJUGATED HYPERBILIRUBINEMIA

Unconjugated hyperbilirubinemia	85% of total should be indirect
In unconjugated hyperbilirubinemia, the fraction of unconjugated bilirubin to total bilirubin exceeds	0.80
Causes of unconjugated hyperbilirubinemia	Hemolytic anemia, Large hematoma, Megaloblastic anemia
Bilirubinemia in Drug Induced hepatitis	Unconjugated bilirubinemia
Bilirubin 4mg in child. Conjugated bilirubin and alkaline	Hemolytic jaundice

phosphatase normal. Bile salts and bile in urine absent. urobilinogen in urine is raised	
Young male with gall stones, serum bilirubin 2.6, Hb6. urine test positive for urobilinogen	Hemolytic jaundice
Unconjugated hyperbilirubinemia, elevated urobilinogen in urine	Hemolytic jaundice

CONJUGATED HYPERBILIRUBINEMIA

Conjugating agent detoxifies bilirubin	Glucoronic acid
Conjugated hyperbilirubinemia is seen in	Stone in common bile duct
Neonatal Hepatitis is	Conjugated Hyperbilirubinemia
Conjugated bilirubin in infancy	Dubin Johnson syndrome, Rotor syndrome, Neonatal hepatitis
Cholestatic jaundice often caused by	Erythromycin estolate
Cholestatic jaundice	Estrogen, Cyclosporine, Phenothiazine
Does NOT cause cholestatic jaundice	INH
Commonest cause of surgical obstructive jaundice	CBD stone
MC Surgical cause of Obstructive jaundice	Choledocholithiasis
Pain in abdomen, jaundice, increased alkaline phosphatase, conjugated hyperbilirubinemia.USG blockade in biliary tree	Clonorchis sinensis
Increased total bilirubin, direct bilirubin, alkaline phosphatase	Obstructive jaundice
70 year old, chest pain, coronary artery disease, USG gall stones, NO past history of biliary colic or jaundice. best treatment	No surgery for gall bladder
Idiopathic cholestatic jaundice	Intense itching, SGOT SGPT less than 60 IU, markedly increased level of alkaline phosphatase
Presence of bilirubin glucoronides in urine with out urobilinogen suggests	Obstructive jaundice
High direct reacting bilirubin and alkaline phosphatase activity pf 85 KA units	Obstructive jaundice
Jaundice in an elderly woman is due to	Stone in CBD
NOT seen in obstructive jaundice	Urobilinogen
NOT a feature of obstructive jaundice	Raised urinary urobilinogen
Increased B12 level NOT seen in	Cholestatic jaundice
Initial Investigation in Obstructive Jaundice	USG
Vitamin deficiency need to be corrected in obstructive jaundice	Vitamin K
Obstructive jaundice with blood loss, replacement with	FFP
Can be done in obstructive jaundice	Vitamin K injection, External drainage
Treatment of conjugated defects in cholestatic jaundice	Concentrate of factors II,VII,IX and X
Element should NOT be given in TPN in Obstructive jaundice	Copper
MC cause of cholestatic jaundice in newborn	Neonatal hepatitis
Jaundice with clay stools, Giant cells on liver biopsy	Neonatal Hepatitis with Extrahepatic biliary atresia

JAUNDICE

Icterus marked in sclera because of	High content of elastin
Breakdown of 1 gm of hemoglobin yields	35 mg of bilirubin
Heme is converted to bilirubin mainly in	Spleen
Bilirubin is derived by	Hemoglobin, Myoglobin
Jaundice at birth or within 24 hours due to	Erythroblastosis
Breast milk jaundice due to	Pregnanediol
Thyroid disorder associated with Prolonged physiological jaundice	Hypothyroidism
Defective hepatic conjugation is seen in	Neonatal jaundice, Gilbert syndrome, Criggler Najjar syndrome
In post hepatic jaundice concentration of conjugated bilirubin in the blood is higher than that of unconjugated bilirubin because	Conjugation process of bilirubin in liver remains operative without interference
Clinical jaundice in neonate	>5 mg/dl
NOT true about physiological jaundice	Conjugated hyperbilirubinemia
Feature of Hepatocellular JAUNDICE	ALT>AST
Charcot's triad ascending cholangitis	Abdominal pain, high fever with rigor, jaundice
Curvoisier Law	Enlarged Non Tender Gallbladder With Jaundice Is Unlikely Gall Stones
Courvoisier law related to	Jaundice
Exceptions to Courvoisier law	*Double impaction, peri ampullary carcinoma, primary oriental cholangiohepatitis causing stones in CBD*
Autosomal dominant familial non hemolytic hyperbilirubinemia	Gilbert disease
Newborn presenting with hyperbilirubinemia	Total and direct bilirubin
Best test to differentiate medical and surgical jaundice	Ultrasound
23 year female, anemia, jaundice, 2 years, peripheral smear showing spheroctes. best investigation	Coomb's test
Most appropriate investigation for 40 year female presenting with anemia, jaundice and spherocytosis	Coomb's test
Metalloproteins help in jaundice by	Inhibit heme oxygenase
Phototherapy is used in	Neonatal jaundice
Indication of Exchange transfusion	Cord bilirubin > 4 mg%, Cord Hemoglobin < 10 mg%, Rate of bilirubin increase > 0.5 mg%
NOT an indication for exchange transfusion	Total bilirubin 10 mg/dL
A 32 weeks premature infant, 900 gm weight on the third day. Serum bilirubin 13 mg%. treatment of choice	Exchange transfusion

CHOLESTASIS

Chlorpromazine	Inflammatory cholestasis
Bland cholestasis or non inflammatory cholestasis	Androgen, Estrogen, OCP, Cyclosporine
Cholestasis is associated with	Erythromycin, Phenothiazines, OCP
Intrahepatic cholestasis	Galactosemia, Cystic fibrosis
Prototype of Intrahepatic Cholestasis	Neonatal Hepatitis

Prototype of Extrahepatic Cholestasis	Biliary Atresia
Neonatal cholestasis seen in	Hepatitis B and C
Byler disease	**Progressive intrahepatic cholestasis I**

CRIGGLER NAJJAR SYNDROME

Crigler Najjar syndrome is due to deficiency of	UDP glucoronyl transferase
Criglar Najjar syndrome is due to defect in	Conjugation
Glucoronyl transferase is absent in	Criggler Najjar type I
NOT true about Crigler Najjar Syndrome I	Liver darkly pigmented
Defect in Criggler Najjar type II	Conjugation of Bilirubin
Crigler Najjar type II syndrome	Recessive

GILBERT SYNDROME

Unconjugated hyperbilirubinemia	Gilbert's syndrome
Well known feature of Gilbert syndrome	*High bilirubin level in prolonged fasting*

DUBIN JOHNSON SYNDROME

Dubin Johnson syndrome	Conjugated hyperbilirubinemia
Dubin Johnson syndrome is due to mutation in	**MRP2**
Melanin like pigmented dark liver	*Dubin Johnson syndrome*
Dubin Johnson syndrome	*Normal life expectancy*
Substance accumulating in Lysosomes in Dubin Johnson syndrome	Epinephrine
Test used to diagnose Dubin Johnson syndrome	Bromosulphaelin test

ROTOR SYNDROME

Rotor syndrome	Conjugated hyperbilirubinemia
Jaundice 50% direct bilirubin.LFT normal	Rotor syndrome

HEMOBILIA

Causes of hemobilia	Trauma to abdomen, Malignancy, Rupture of hepatic artery aneurysm
MC cause of hemobilia	Trauma
NOT a cause of hemobilia	Hepatitis
Abdominal pain, jaundice and malena	Hemobilia
NOT a feature of hemobilia	Shock, Fever

REYE SYNDROME

Jamshedpur fever	*Reye syndrome*
Reye syndrome is caused by	Adenovirus, Herpes, Influenza
Reye syndrome	Frequently complicated by viral infections, Prothrombin is prolonged, Precipitated by salicylates, Moderate elevation of SGOT and SGPT, Hypoglycemia, Hypoglycorrhoea
Reye syndrome	Cerebral edema, Microfatty change in liver without inflammatory changes
Reye Syndrome	Hypoglycemia, Raised Ammonia, Increased Prothrombin time, Cerebral edema, Microfatty changes in Liver without inflammatory changes
Reye syndrome is associated with	*Microvesicular fatty change*
A child presents with metabolic lactic acidosis along with hyperammonemia and convulsion is due to	Reye syndrome
A young 6 yrs old child, hepatitis, cerebral edema. What will be liver biopsy finding?	Microvesicular steatosis
MC prodromal symptom of Reye syndrome	Vomiting
NOT true about Reye's syndrome	Seen with ampicillin therapy
NOT seen in Reye syndrome	Increased serum bilirubin
Histological finding in Reye syndrome	Glycogen depletion

FATTY LIVER

Macrovesicular fatty liver	Protein energy malnutrition

MIRIZZI SYNDROME

MIRIZZI SYNDROME	Obstructive Jaundice, Recurrent Ascending Cholangitis, Gallstones in Hartman's Pouch externally compressing common bile duct
Mirizzi syndrome shows symptoms of	*Stone ulcerating into common bile duct*
Mirizzi syndrome	Stone compressing gall bladder

GENERAL FEATURES OF HEPATITIS

Asiatic cholangiohepatitis is caused by	Clonorchis sinensis
Giant cell neonatal hepatitis due to	α1- antitrypsin deficiency
Granulomatous hepatitis	Carbamazepine, allopurinol, phenylbutazone
Oxacillin is associated with	Hepatitis
In non hemolytic jaundice, urobilinogen is seen in	Infective hepatitis
Average incubation period of infectious hepatitis	25 days
Pain upper abdomen, nausea, decreased appetite, undergone cholecystectomy 2 years back. bilirubin increased, SGOT 900 SGPT 700 serum alkaline	Acute viral hepatitis

phosphatase 280	
Councilman bodies	Acute viral hepatitis
Features of Acute Viral hepatitis	Councilman bodies, Ballooning degeneration, Hepatic cell necrosis
Sexually transmitted hepatitis	*Hepatitis A, B, D*
Super infection and co infection are seen between	HBV & HDV
MC pathological change in acute viral hepatitis	Ballooning degeneration
NOT a biopsy feature of acute viral hepatitis	Ground glass hepatocytes
NOT correct regarding hepatitis management	Antiviral drugs-acute viral hepatitis
Anti Liver Kidney Microsomal 1 (LKM- 1)	Chronic Hepatitis C, Autoimmune Hepatitis
Anti Liver Kidney Microsomal 2 (LKM- 2)	Drug Induced Hepatitis
Anti Liver Kidney Microsomal 3(LKM- 3)	Chronic Hepatitis D
Vaccines available for	Hepatitis A and Hepatitis B

HEPATITIS A

Enterovirus E72 is	Hepatitis A virus
Hepatitis A virus belongs to	Picorna virus
MC cause of Sporadic Hepatitis in Children	Hepatitis A
Fecoorally transmitted	Hepatitis A
Only hepatitis virus which can be culture	Hepatitis A
Cultivable hepatitis virus	Hepatitis A
Transmission of Hepatitis A occurs	2 weeks before and 1 week after onset of symptoms
Incubation period of Hepatitis A	15-45 days
Feature of Hepatitis A	Cause mild illness in children
Hepatitis A virus shedding in feces	2 weeks before symptoms and 2 weeks therafter
Isolation period of hepatitis A	3 weeks
NOT a sexually transmitted disease	Hepatitis A
Hepatitis A is best demonstrated by	Immunomicroscopy
Hepatitis A virus is NOT destroyed by	1:4000 formalin, UV radiation, Boiling at 100 degree Celsius for 5 minutes

FEATURES OF HEPATITIS B

Hepatitis B is	Double stranded DNA virus
Dane particle	HBV
Dane particle refers to	Complete HBV
Dane particle contains	Surface antigen and core antigen
Hepatitis B belongs to	Hepadna virus
HBV prevalent in North & West India	Awy(B)
HBV prevalent in south & East India	Adr(C)
Reverse trancriptase of hepatitis B virus is coded on	P gene
Largest genome of Hepatitis	P gene (DNA polymerase)
Incubation period of Hepatitis B	4 – 24 weeks, 45-180 days
Median incubation period of hepatitis B	*100 days*
Incubation period of hepatitis B virus	6 weeks to 6 months
MC cause of Hepatitis B in 3 year old Child	Blood Transfusion
MC Hepatitis Virus transmitted perinatally	HBV

Highest risk of fetal transmission in first trimester	Hepatitis B
MC time of perinatal transmission of HBV	Time of delivery
Significant perinatal transmitted hepatitis	Hepatitis B
Highest transmission of hepatitis B from mother to fetus occurs if the mother is infected during	III trimester
Hepatitis B can be transmitted by	Semen, blood, breast milk
Chances of vertical transmission of hepatitis B can be as high as	90%
NOT true about hepatitis B	Vertical transmission more important than horizontal
Hepatitis B virus NOT associated with	Cholangiocarcinoma
Does NOT infect CNS	Hepatitis B
Organism associated with manifestation of immune complex disease	*Hepatitis B*
A patient has Bilirubin =0.9 mg/dl, elevated SGPT & SGOT (~1500-1600 IU/L). HBsAg positive, Anti HBC IgMnegative, Anti Hepatitis E IgM positive. Patient has	Chronic Hepatitis B infection with Hepatitis E superinfection
Best prognosis with HBV Infection	Acute HBV Infection
Worst Prognosis in HBV Infection	HDV Superinfection of HBV
Councilman Bodies	Hepatitis B
MC Presentation of Hepatitis in Neonates	Asymptomatic HbsAg Carrier
Hepatitis having poor prognosis	Hepatitis B
Ground glass hepatitis	*Hepatitis B*
Ground glass granular eosinophilic cytoplasm with sanded nuclei	*Hepatitis B*
Extrahepatic manifestations of hepatitis B	Aplastic anemia, Guillain barre syndrome, Pancreatitis, Cryoglobulinemia
Glomerulonephritis during hepatitis B infection	Membranous glomerulonephritis, Membranoproliferative glomerulonephritis, Mesangiocapillary glomerulonephritis
HBV is NOT associated with	Hepatic adenoma
Steatosis is NOT seen in	Hepatitis B infection
NOT true about HepB	Antiviral used in management of acute HepB

DIAGNOSIS OF HEPATITIS B

Australian antigen	*HbsAg*
HbsAg may be present in	Saliva
Earliest marker of acute Hepatitis B	HbsAg
Epidemiological marker of hepatitis B	HbsAg
Virological marker indicating first evidence of hepatitis B infection	HbsAg
Marked increase in HbsAg	Chronic active hepatitis
Early diagnosis of acute hepatitis B	Presence of HbsAg in serum
HbsAg DOES NOT indicate	Severity of disease
Indicator of active multiplication of hepatitis B virus	HbeAg
Most Important factor determining Perinatal transmission of Hepatitis	HbeAg
Marker in window period	Anti Hbc
Epidemiological marker for HBV	*Anti HBC*

Diagnostic marker of acute Hepatitis B	IgM anti Hbc
Investigations done if acute viral hepatitis is suspected	HbsAg, IgM anti Hbc, IgM anti HAV, Anti HCV
Recent hepatitis B/acute hepatitis B infection diagnosed by	IgM anti Hbc ab
Hepatitis B seroconversion starts with	IgM anti HbC
Antibody present during window period of hepatitis	IgM anti HBc
Acute infection with HBV is characterized by	IgM anti HBcAg and HBsAg
HbsAg, IgM anti Hbc, HbeAg	Acute hepatitis B with high infectivity
Blood donor is NOT considered for safe transfusion, if he has	HBsAg positive, IgM anti HBc positive
Antigen NOT appearing in blood	HbcAg
Hepatitis B infectivity indicated by	HbsAg and HbeAg
Child HbsAg positive. Mother carrier. Mother's profile	HbsAg and HbeAg positivity
Marker of acute hepatitis B infection	DNA polymerase
Traditional Marker for HBV Replication	HBV DNA Polymerase
Markers of active hepatitis or persistent replication	HBV DNA, HBV DNA polymerase, HbeAg
Marker for Hepatitis B vaccination	Anti HbsAg
Child after hepatitis B vaccination	Obtain anti HbsAg levels
Anti HbsAg positivity indicates	Immunisation
30 year female, healthy baby 37 weeks of gestation. Known case of hepatitis B infection. Positive for HbsAg and negative for HbeAg. most appropriate treatment for baby	Both active and passive immunization soon after birth
HbsAg positive, asymptomatic others normal	HBV carrier
HBsAg positive, HbeAg negative, Anti HBeAg antibody positive, HBV DNA copies are absorbed to be 1 lakh per ml, SGOT, SGPT elevated to 6 times	HBV precore mutant
Precore mutant chronic hepatitis B	Alteration in precore regions rendering virus incapable of encoding HbeAg
Precore mutant chronic hepatitis B	Severe chronic hepatitis infection with detectable HBV DNA levels, HbeAg typically negative
35 year oldman found positive for HBsAg and HBeAg, accidentally during blood donation. SGOT SGPT normal. next investigation	HBV DNA estimation
Young male found to be HbsAg positive and HbeAg negative. Liver enzymes were normal. next step in management of young male	Serial monitoring
Serology of a young man shows HbsAg, however HbeAg is negative with normal levels of AST and ALT. He is asymptomatic. what is the next line of management	Wait and watch
HbeAg negative individuals with ALT less than or equal to upper limit of normal	Do not require active antiviral treatment

TREATMENT OF HEPATITIS B

Treatment of acute viral hepatitis	Supportive
Drugs used for management of HBV	**Adefovir, Entecavir, Telbivudine, Tenofovir, Pegylated interferon, Lamivudine**
Optimum treatment inpatient with chronic hepatitis B with raised AST levels	Lamivudine

Treatment of Choice for Hepatitis B	Interferon α + Lamivudine
Lamivudine is recommended for treatment of chronic hepatitis B when	ALT > twice normal upper limit
Agents used for treatment of chronic hepatitis B	Interferon, Lamivudine, Adefovir, Entecevir
Drug that can cause histopathological resolution in hepatitis B	Adefovir
Interferon treatment is recommended in hepatitis patient with	Increased HBV DNA and increased ALT
Pegylated interferon in NOT used in	**Cirrhosis, Transplantation, Immunocompromised**

FEATURES OF HEPATITIS C

Hepatitis C belongs to	Flavi virus
Feature of HCV	Enveloped RNA virus
MC genotype of HCV	**Type I**
MC cause of Chronic Hepatitis	Hepatitis C
MC Indication of Liver Transplantation	Chronic Hepatitis C
Transmission of Hepatitis C virus	Parenteral
Hepatitis C virus transmission during pregnancy depends on	*Route of delivery*
Hepatitis C is associated with	Antibody to HCV may not be seen in acute stage
Arthritis, purpura, monoclonal polyclonal cryoglobulins, deposits of cryoglobulin around vessels. patient should be tested for	HCV
Extrahepatic manifestations of hepatitis C	Lichen planus, Glomerulonephritis, Cryoglobulinemia, Polyarthritis, PAN
Chance of hepatitis type C becoming chronic infection	>50%
HCV screening	Medical students are screened before their joining, IV drug abuser are prone to infection, Long term hemodialysis, Interferon is treatment
Antibodies for HCV more than 6 months, no signs, AST/ALT normal	Determine HCV RNA levels
Most sensitive indicator of infection with hepatitis C virus	*HCV RNA*
Antibodies to HCV may NOT be seen in	*Acute stage*
Episodic transfer of aminotransferase in seen in	*Hepatitis C*
HPE features of hepatitis C infection	*Cholangiole formation*

MANAGEMENT OF HEPATITIS C

Recombinant immunoblast is for	*HCV*
Chronic hepatitis C responded to interferon. however one year after relapse	Ribavirin and interferon
Treatment of Choice for Hepatitis C	Interferon α + Ribavirin
Treatment of Hepatitis C	Pegylated Interferon + Ribavirin

Sustained response to antiviral therapy with interferon and ribavirin in hepatitis C is indicated by	Female sex

HEPATITIS D

Hepatitis D virus resemble	Some plant virus

HEPATITIS E

Calcivirus	Hepatitis E
Hepatitis E clinically resembles	Hepatitis A
HEV clinically resembles	HBV
MC route of spread in hepatitis E	Fecooral
NOT transmitted through sexual route	Hepatitis E
MC cause of Fulminant Hepatitis in Pregnancy	Hepatitis E
MC type of hepatitis responsible for epidemics in India	Hepatitis E
MC cause of Hepatic Encephalopathy in Pregnancy	Hepatitis E
MC cause of Epidemic Hepatitis	Hepatitis E
MC cause of Sporadic Hepatitis in Adults	Hepatitis E
Fulminant hepatic failure in pregnant woman	Hepatitis E
Non parenteral hepatitis	Hepatitis E
Which does not go into Chronic hepatitis stage	Hepatitis E
During an epidemic of hepatitis E, fatality is maximum in	Pregnant woman

HEPATITIS G VIRUS

Hepatitis G virus	*Also known as GB virus, blood borne RNA virus*

CHRONIC HEPATITIS

Infection leading to chronic carrier state	HCV, HBV
Carrier state does NOT exist for	*HAV*
MC symptom of chronic hepatitis C	**Fatigue**
Features of ongoing activity of Chronic hepatitis	Bile stasis, Fibrosis, Necrosis of Hepatocytes
In chronic viral hepatitis	Grading refers to extent of necrosis and inflammation
Piecemeal necrosis is seen in	Chronic active hepatitis
Piecemeal necrosis	*Destruction of the limiting plate with necrosis of hepatocytes surrounding portal triad*
Chronic hepatitis B is associated with	*Polyarteritis nodosa*
Chronic active hepatitis differentiated from chronic persistent hepatitis by	Histopathology
Chronic active hepatitis	*HLA DR3*

NOT seen in chronic active hepatitis	IgM against core antigen
HAI (histological activity index) and METAVIR for	**Chronic hepatitis**
Best prognostic factor in chronic hepatitis C	**Liver histology**
Treatment of chronic viral hepatitis	Antiviral therapy

ALCOHOLIC HEPATITIS

Alcoholic hepatitis	Gamma gluatmyl transferase raised, SGOT> SGPT, Alkaline phosphatase is raised
Features of alcoholic hepatitis	Elevated bilirubin, Prolonged prothrombin time, Anemia, HYPOalbuminemia
Alcoholic liver injury	*Steatosis, Mallory hyaline, centrilobular fibrosis, lobular inflammatory infiltrate with a conspicuous component of neutrophils*
Mallory Bodies	Alcoholic Hepatitis
Candle dropping is seen in	*Alcoholic hepatitis*
Ratio of AST/ALT>1	Alcoholic hepatitis
Fatty change in liver is characteristically seen in	Chronic alcoholism
NOT seen in liver of chronic alcoholic	Granuloma formation
Alcoholic hyaline in alcoholic liver disease is composed of	Eosinophilic intracytoplasmic inclusions
Pigment deposited in hepatocytes in alcoholic liver disease	Hemosiderin
Enzyme used to diagnose alcoholism	SGOT/AST
Drugs used for treatment of alcoholic hepatitis	**Prednisolone, Pentoxifylline**
Drug shown to reduce mortality and renal failure in acute liver disease due to alcoholism	Pentoxifylline

AUTOIMMUNE HEPATITIS

Following liver transplantation, recurrence of primary disease in liver most likely occur in	Autoimmune hepatitis
Characteristic antibodies in autoimmune hepatitis	ANA, Anti SLA, anti LKM1
A male with right upper quadrant pain jaundice treatment successful with azathioprine	Autoimmune Hepatitis

NON ALCOHOLIC FATTY LIVER DISEASE

MC cause of Cryptogenic Cirrhosis	Non Alcoholic Fatty Liver Disease
Non alcoholic steatohepatitis	Diabetes, Obesity, Hypertrigylceridemia, Total parenteral nutrition
NASH	Common in diabetes, Elevated transaminases, Clofibrate beneficial
A diabetic female, jaundice, pathological finding	NASH

ASCITES

Ascites more than leg edema	Constrictive pericarditis, tricuspid valve lesion, liver pathology
Snowstorm ascites	Meconium ileus
Chylous ascites	Trauma, tuberculosis, nephrotic syndrome
Chylous ascites NOT seen in	Colloid carcinoma of stomach
Pseudochylous ascites	Malignant ascites
Hemorrhagic ascites is diagnosed when RBC count >	10,000 /mm3
SAAG > 1.1 g/dl means	**Ascites is due to portal hypertension**
True about ascites	USG can detect as little as 100 ml of peritoneal fluid
First line treatment in ascites	Salt restriction
Drug used for ascites	Spironolactone

FEATURES OF HEPATIC FAILURE

MC site of metastatic disease	Liver
Centrilobular necrosis of liver occurs with	Phenol, carbon tetrachloride
Centrilobular necrosis of liver	CCl2
Central lobular necrosis in Liver is due to	Hemorrhagic shock, Chronic Venous congestion, Halothane
First laboratory test becoming abnormal in hepatocellular failure	Prothrombin time
Features of acute fulminant hepatic failure	Hypoglycemia, Hypomagnesemia, Hepatorenal syndrome, Intracranial hemorrhage, Coagulopathy
Lower than normal in severe liver failure	*Urea*
Best prognostic indicator in acute liver failure	Factor V
Most Important Prognostic factor in Children with Acute Liver Failure	Increased Prothrombin Time
Best prognostic factor in acute liver disease	Factor V
NOT included in MELD score	Albumin
NOT a criteria in Child Pugh's Classification	Alkaline Phosphatase
Stigmata of chronic liver disease	Palmar erythema, Spider naevi, Testicular atrophy, Paper money skin, Drum stick finger
Vasodilatation in spider nevi due to	Estrogen
Orthodeoxia is a feature of	*Hepatopulmonary syndrome*
Histopathological finding in liver in malaria	Kupffer cell hyperplasia with macrophage infiltration around periportal area laden with pigments
Hallmark of irreversible liver damage	Periportal fibrosis
Granuloma in liver is caused by	Allopurinol
Liver granulation may be associated with	Candida, halothane, sarcoidosis
Liver granuloma is NOT associated with	Hepatic metastasis, Chronic alcoholism
Flapping tremor	Hepatic encephalopathy, Uremia, CO2 narcosis
Flapping tremor NOT seen in	Thyrotoxicosis
Helps in differentiating acute and chronic liver disease	Albumin
d-dimer in liver disease is	Normal
More specific indicator for liver injury	ALT
Decrease mortality and renal failure in acute liver	Pentoxyfylline

disease due to alcoholism	

MANAGEMENT OF HEPATIC FAILURE

Quantitative estimation of liver function	Galactose elimination capacity

HEPATORENAL SYNDROME

Zellweger syndrome	*Cerebral hepatorenal syndrome*
Hepatorenal syndrome	Urinary sodium is less than 10 mmol/L, urine osmolality is greater than plasma osmolality, poor response to volume expansion, Normal renal histology, Creatinine clearance less than 40 ml/min, No sustained improvement in renal function after volume expansion
Treatment of hepatorenal syndrome	**Terlipressin, Octreotide, Midodrine, Norepinephrine, IV albumin**
Treatment of choice for hepatorenal syndrome	**Orthotopic liver transplantation**

FEATURES OF HEPATOCELLULAR CARCINOMA

Most Important Risk factor for Hepatocellular Carcinoma	Cirrhosis
Primary hepatocellular carcinoma common in	Asians
Vinyl chloride has been implicated in	Angiosarcoma of Liver
Mutation of β- cantenin	Hepatoblastoma, Hepatocellular Carcinoma
NOT a risk factor for hepatocellular carcinoma	Animal fat in diet
MC Primary Hepatic Malignancy	Hepatocellular Carcinoma
Classification of hepatocellular carcinoma	**CLIP classification, Oguda classification**
Hepatocellular carcinoma	AFP is increased in 70% of cases, Resection possible in few cases only, USG guided aspiration biopsy is used for diagnosis
38 year old man chronic alcoholic pain in abdomen.liver is enlarged serum alpha fetoprotein is elevated	Hepatocellular carcinoma
40 year male alcoholic, lump in right lobe of liver, AFP normal	Hepatocellular carcinoma
Liver tumour that may invade portal vein or hepatic vein	Hepatocellular carcinoma
Strong propensity for vascular invasion is seen in	Hepatocellular carcinoma
Maximum increase in alpha fetoprotein is seen in	Hepatocellular carcinoma
Serum AFP greater than 10 times the normal limit, common cause	Metastatic carcinoma of liver
Marker for Hepatocellular carcinoma	Alpha fetoprotein
Most Unlikely feature of Hepatocellular Carcinoma	Jaundice
NOT true about hepatocellular carcinoma	Over 80% of tumours are surgically resectable
NOT a paraneoplastic syndrome for hepatocellular carcinoma	*Hyperglycemia*

FIBROLAMELLAR VARIANT OF HEPATOCELLULAR CARCINOMA

Fibrolamellar variant	Hard scirrhous tumour, Young males and females, Good prognosis
Fibrolamellar type	Good prognosis, Young females
NOT true about fibrolamellar carcinoma of liver	AFP always greater than 1000, Hepatitis B is an important risk factor

DIAGNOSIS OF HEPATOCELLULAR CARCINOMA

Serological assay for hepatocellular carcinoma	**AFP – L3, des gamma carboxy prothrombin**
Tumor marker for primary hepatocellular cancer	Alpha fetoprotein, PIVKA 2, neurotensin
Alpha fetoprotein	No AFP elevation in fibrolamellar variant, Preoperative high level indicate worst prognosis, High level seen in stomach carcinoma, Levels may be increased in hepatitis
NOT a tumour marker for primary hepatocellular carcinoma	Alpha 2 macroglobulin

TREATMENT OF HEPATOCELLULAR CARCINOMA

Criteria for surgery for hepatocellular carcinoma	**Milian**
Unaffected tissue margin resected in hepatocellular carcinoma	*1 – 2 cm*
Transarterial chemoembolization is used in treatment of	*Liver tumor*
NOT a treatment modality of hepatocellular carcinoma	Nd Yag laser ablation
NOT used for in situ ablation of liver secondaries	Ultrasonic waves
NOT a novel treatment of hepatocellular carcinoma	*Mantle radiation therapy*

WILSON'S DISEASE

Increased copper excretion in urine is seen in	Primary sclerosing cholangitis, Wilson's disease, Primary biliary cirrhosis
Increased copper excretion in urine is NOT seen in	Hepatocellular carcinoma
Gene associated with Wilson's disease	ATP7B
Wilson's Disease (ATP7B)	Chromsosome 13
Congenital Wilson disease	May present as acute hepatitis
Sunflower Cataract	Wilson's disease, Chalcosis, Penetrating trauma
Behavioural symptoms, extrapyramidal symptoms, liver damage	Wilson's disease
TRUE statement about Wilson's disease	Hemolytic Anaemia
Ocular Manifestation of Wilson's disease	KF ring
Kayser Fleischer ring in cornea is due to	Copper

KF ring is due to deposition of copper in	Descement's membrane
KF ring seen in	Wilson's disease
KF ring	Characteristically seen in Wilson's disease, but NOT pathogonomic, does NOT affect vision
Wing beating tremor	*Wilson's disease*
NOT true about Wilson disease	Urinary copper excretion < 100 microgram/day
NOT seen in Wilson's disease	Peripheral neuropathy, testicular atrophy
NOT seen in Wilson's Disease	Sensory Changes
NOT true regarding Wilson's disease	Increased Serum Ceruloplasmin level
Prognosis of Wilson's disease	**Nazer prognostic index**
NOT included in Nazer prognostic index	**Alkaline phosphatase**
NOT included in Nazer prognostic index	*Serum albumin*
Diagnostic feature of Wilson disease	Low serum ceruloplasmin and high urinary copper
Investigation for Wilson's Disease	Liver Biopsy
Face of giant panda sign on MRI	*Wilson's disease*
Drugs used in management of Wilson's disease	**Penicillamine + pyridoxine, Trientine, zinc**
Initial neurologic therapy for Wilson's disease	**Tetrathiomolybdate**
Maintenance in Wilson disease	Zinc acetate
Drug of choice for patients with hepatitis or cirrhosis without evidence of hepatic decompensation and neuropsychiatric symptoms	Zinc
Trientine	NOT potent than penicillamine, Alternate to penicillamine in non tolerant patients, Should NOT be administered within 2 hours of iron supplementation, Can cause iron deficiency anemia which is reversible by oral iron supplements
Anti copper therapy in Wilson's disease for	*Life long*

HEMOCHROMATOSIS

Hepcidin is involved in	**Iron metabolism**
Inheritance of Hemochromatosis	Autosomal recessive
Hemochromatosis	Genetically heterogenous
MC HPE mutation in hemochromatosis	*C28LY*
HLA associated with primary hemochromatosis	*HLA A3*
Bronze diabetes	Hemochromatosis
Weakness, pain in upper abdomen, hyperpigmentation, arthritis, hyperglycemia, enlarged palpable liver	Hemochromatosis
Arthritis, hyperpigmentation of skin, hypogonadotrophic hypogonadism	Hemochromatosis
Skin pigmentation in hemochromatosis is due to	Melanin
Hemochromatosis associated with	Diabetes, Cirrhosis, CHF
MC cardiac manifestation of hemochromatosis	**Congestive cardiac failure**
Hemosiderin deposition in Hemochromatosis	Islet cells, Acinar cells, Interstitial stroma
Hemosiderin is stained by	Prussian blue
Secondary hemochromatosis is associated with	Thalassemia, Sideroblastic anemia, Multiple drug transfusions

Hemochromatosis does NOT affect	Salivary gland
Elevated serum ferritin, serum iron, percent transferrin saturation	Hemochromatosis
Early phenotypic manifestation of idiopathic hereditary hemochromatosis	Increased transferrin saturation
High transferrin levels are associated with	*Homozygous form*
54 year male, known diabetic develop cirrhosis, skin hyperpigmentation and restrictive cardiomyopathy. best initial investigation	Iron binding capacity
SQUID measure	*Heptic iron*
HPE finding in hemochromatosis	*Prussian blue positive*
A child history of 8 blood transfusions in 2months Hb- 6, MCV low, WBC is normal, TLC 5600, also hypochromic microcytic anemia, which investigation is not done next?	Urinary hemosiderin
Treatment of Choice for Hemochromatosis	Phlebotomy
Bone marrow transplant NOT a treatment for	Hemochromatosis
NOT true about hereditary hemochromatosis	Desferroxamine is the treatment of choice

HYDATID CYST OF LIVER

Hydatid cyst of liver	Caused by Echinococcus granulosus, In 80% of cases its single, Mostly asymptomatic, Most common in right lobe of liver, Laparoscopic aspiration of cyst is performed
A 40 year old male presents with painless cystic liver enlargement of 4 years duration without fever of jaundice	Hydatid cyst of liver
Crumbled egg appearace of liver	Hydatid cyst
Gharbi classification for	*Hydatid cyst of liver (stage V calcified)*
NOT a complication of hydatid cyst in liver	Cirrhosis
Treatment of echinococcus granuosus	Albendazole
Hydatid disease	20% saline is scolicidal, PAIR is done in hepatid hydatids
PAIR is contraindicated in	Lung abscess, Inaccessible location
PAIR is contraindicated in	*Superficially located cysts, cysts with multiple thick internal septal division, cysts communicating with biliary tree*
NOT a scolicidal agent	Povidone iodine

PYOGENIC LIVER ABSCESS

MC cause of pyogenic liver abscess	Hematogenous spread from distant site, Biliary sepsis, Ascending infection through biliary tract
MC cause of pyogenic liver abscess	Streptococcus milleri
MC presenting sign of liver abscess	**Fever**
Pyogenic liver absess	Single and large abscess
Indication of aspiration in liver abscess	Left lobe, >10 cm size

AMOEBIC LIVER ABSCESS

Amoebic liver abscess	Most common in right liver lobe, Can rupture into pleural cavity
MC site of amoebic liver abscess	Right lobe
Anchovy sauce pus	Amoebic liver abscess
NOT true about amoebic liver abscess	Common in female
Treatment of Hepatic amoebiasis	Mostly treated conservatively
Treatment of amoebic liver abscess	**Tinidazole, Metronidazole**
NOT used in treatment of amoebic liver abscess	Diloxanide furoate

HEPATIC ENCEPHALOPATHY

Substance incriminated in pathogenesis of Hepatic encephalopathy	Ammonia
Hepatic encephalopathy precipitated by	Anemia, barbiturates, hypothyroidism
Precipitating factor for hepatic encephalopathy in chronic liver disease	Hypokalemia, hyponatremia, hypoxia. metabolic alkalosis
Hepatic encephalopathy predisposed by	Dehydration, Constipation, GI bleeding, SBP
Symmetric high voltage, triphasic slow wave pattern	Hepatic encephalopathy
Foetur Hepaticus	Hepatic Encephalopathy
Asterixis	Respiratory failure, uraemia, decompensated hepatic failure
Unilateral asterixis	Acute Parietal Lesion
Drug used for gut sterilization in patient with hepatic encephalopathy	Neomycin
Useful in hepatic encephalopathy	lactulose
Common side effect after TIPPS	Hepatic Encephalopathy

LIVER TRANSPLANTATION

Liver transplantation first done by	Starzl
Most important factor in liver transplantation	**Timing**
King's criteria for orthoptic liver transplantation for non paracetamol induced acute liver failure	*Age <10 or >40, etiology is non A, non B, idiosyncratic or halothane induced, more than 7 days jaundice before encephalopathy, PT>50 seconds, bilirubin >300 micromols/L - any 3 of the above or PT > 100 seconds*
Indications of liver transplantation	Hemochromatosis, Primary bilary cirrhosis, Sclerosing cholangitis
MC Indication of Liver Transplantation	Cirrhosis
Commonest indication of liver transplantation in infant	Biliary atresia
NOT an indication of liver transplantation	Hepatitis A, HIV
Liver transplantation is NOT indicated in	Metastasis to Liver

Auxiliary Orthoptic Liver Transplantation (Donor Liver not removed, Recipient liver placed)	Acute Fulminant Liver Failure
Auxiliary orthoptic liver transplantation is indicated for	Acute fulminant liver failure for any cause
In orthotopic liver transplantation, best way to get bile drainage in donor liver	Anastomose donor bile duct with recipient bile duct or Roux en Y choledochojejunostomy
Best way to get bile drainage in donor liver in orthoptic liver transplantation	Donor bile duct with recipient bile duct or Roux en Y choledochojejunostomy
Treatment of post transplant sinusoidal obstruction syndrome of liver	*Difibrotide (polydeoxyradionucleotide)*

CIRRHOSIS

Bridging fibrosis and nodularity	**Stage 3**
Cirrhosis	**Stage 4**
MC cause of Cirrhosis	Alcoholic Liver Disease
Cause of cirrhosis in childhood	Alpha 1 antitrypsin deficiency
MC genetic cause of liver disease in children	Alpha 1 antitrypsin deficiency
Drug causing cirrhosis of liver	Methotrexate
Site of obstruction in cirrhosis	Sinusoids
Major source of collagen in Cirrhosis	Ito cells (Hepatic stellate cells)
In cirrhosis, collagen is laid down by	Hepatic stellate cells
In advanced stages of cirrhosis, major blood supply of liver from	Hepatic artery
Alcoholic cirrhosis is associated with	**Palmar erythema**
Increased in hepatic cirrhosis	Gamma globulins
Spontaneous bacterial peritonitis occurs in	Cirrhosis
MC cause of spontaneous bacterial peritonitis	**E.coli**
Cirrhosis of Liver with best prognosis	Hemochromatosis
Cardiac cirrhosis	Nutmeg cells
Micronodular cirrhosis	Alcoholic liver disease, Hemochromatosis, Chronic extrahepatic biliary obstruction, Indian childhood cirrhosis, Wilson's disease
Micronodular cirrhosis NOT seen in	Chronic hepatitis B, Budd chiari syndrome
Macronodular cirrhosis	Chronic hepatitis B
Macronodular cirrhosis is seen in	Post necrotic
Post necrotic cirrhosis	Decreased liver span, Shrunken liver
NOT seen in Indian childhood cirrhosis	Fatty change
Child criteria for	Cirrhosis
NOT a part of child pugh criteria	Nutrition
In child's criteria partial encephalopathy, bilirubin 2.5 mg/dl, albumin 3 mg/dl, controlled ascites	Grade B
In cirrhosis of liver which drug is contraindicated?	Acetazolamide
Does NOT require dose reduction in cirrhosis	Lorazepam
Child pugh moderate to severe hepatic insufficiency managed by	Orthoptic liver transplantation

BILIARY CIRRHOSIS

Untreated cases of bile duct injury develop	Secondary biliary cirrhosis
Primary Biliary cirrhosis	Inflammation of Intrahepatic bile duct
Primary biliary cirrhosis	Increased risk of hepatocellular carcinoma, Often asymptomatic, Elevated IgM, Positive antimitochondrial antibody
Primary biliary cirrhosis	*Common in females, anti smooth muscle antibodies are diagnostic, portal hypertension is a known complication*
MC symptom of Primary biliary cirrhosis	Pruritis
Most important features of primary biliary cirrhosis	Generalized pruritis, Fatigue
Anti mitochondrial antibody	Primary Biliary Cirrhosis
Anti p62, Anti sp 100, Anti glycoprotein 210	Primary Biliary Cirrhosis
Feature of Primary biliary cirrhosis	Elevated lipoprotein X

NON CIRRHOTIC PORTAL FIBROSIS

Arsenic ingestion cause	Non cirrhotic portal fibrosis
MC presenting symptom for Non cirrhotic portal hypertension	Upper GI bleeding
Esophageal varices with no heptomegaly, no ascites	Non cirrhotic portal fibrosis
8 year boy, upper GI bleeding, splenomegaly, no signs of ascites or hepatomegaly. esophageal varices are found on UGIE	Non cirrhotic portal fibrosis
Massive hematemesis, systemic hypotension, no fever, massive splenomegaly and No hepatomegaly	Non cirrhotic portal fibrosis
NOT a feature of non cirrhotic portal fibrosis	Bridging fibrosis

PORTAL HYPERTENSION

Normal portal vein pressure	5-10 mm Hg
Structure carrying blood from esophagus to Portal Vein	Left Gastric Vein
Portocaval anastomosis is seen between	In the rectum, superior rectal vein and inferior rectal vein
Portal systemic anastomotic vein does not include	Left renal vein
Portal and Systemic circulation does NOT anastomose at	Pyloric end of Stomach
Drug induced portal hypertension seen with	Vitamin A toxicity
Arsenic cause	Basal cell carcinoma, portal hypertension
MC cause of Portal Hypertension in Adults USA	Cirrhosis, 2nd Portal Vein Obstruction
MC cause of Portal Hypertension in Indian adult	Non Cirrhotic Portal Fibrosis
MC cause of Portal Hypertension in Children	Extrahepatic portal Vein Obstruction
Portal hypertension in children in India is commonly due to	Extrahepatic portal vein obstruction
MC cause of severe hematemesis in child	Portal hypertension
7 year girl, massive hematemesis and malena, large	Portal hypertension due to extraheptic obstruction

spleen ,non palpable liver, mild ascites, portal vein NOT visualized in USG,LFT normal. Endoscopy revealed esophageal varices	
Commonest site of varices in portal hypertension	Esophageal
Most constant finding in patient with Portal Hypertension	Splenomegaly
Bleeding esophageal varices commonly occurs in	Portal hypertension
On barium swallow X-ray of esophagus reveals esophageal varices due to	Portal hypertension
Internal hemorrhoids are often manifestation of	Portal hypertension
50 year male hematemesis, 500 ml of blood.90/60 mm Hg.110/min. splenomegaly 5 cm below costal margin	Portal hypertension
Portal hypertension following portal vein thrombosis are guided by	Increase in splenic pulp pressure, Increase in portal vein pressure, Portal vein Doppler study
TIPPS between	Portal and hepatic vein
TIPS	Type of portocaval shunt, Intrahepatic shunt, Most suitable for patient going for liver transplantation
TIPS is contraindicated in	Post shunt encephalopathy
Long term complication of TIPS	*Stenosis*
Portocaval shunt in portal hypertension	Left splenic vein to Left renal vein
Portosystemic shunt NOT seen in	Spleen
Contraindications of portal systemic shunting	Serum albumin less than 3 mg%, Massive ascites, Significant jaundice
Prognosis of portocaval shunt does NOT depend of	Type of shunt
Treatment of choice in 45 year old patient presented with severe hematemesis	Whole blood transfusion
Treatment of Left Sided Portal Hypertension	Splenectomy

ESOPHAGEAL VARICES

Esophageal varices drain in to	*Left gastric and hemiazygous vein*
Sites of bleeding in gastroesophageal varices	Short gastric vein, Right gastric vein, Left gastric vein
MC Presentation of Esophageal Varices	Massive Hematemesis
18 year old, massive hematemesis, history of fever for past 14 days, managed with drugs moderate splenomegaly	Esophageal varices
Young patient, massive hematemesis and splenomegaly. Source of bleeding	Esophageal varices
A child is brought by mother with history of massive hematemesis drug intake previously with NSAIDs and on treatment.associated with moderate splenomegaly	Esophageal varices
Grade IV Esophageal varices in barium swallow	Mucosal folds below carina
A vasopressin analogue does NOT produce therapeutic effect through vasopressin V2 receptor in	Bleeding esophageal varices
Bleeding tendency in case of esophageal varices depend on	Size and site of varices
Management of Bleeding Varices in pregnancy	Observation
Useful in medical treatment of variceal bleeding	Octreotide
Octreotide	Suppresses growth hormone secretion, useful for variceal bleeding, secretory diarrhea, NOT active orally

NOT used for variceal bleeding	Desmopressin
Pressure of Senstaken tube to control varices	35 mm Hg
Sengstaken tube should be deflated temporarily after	*12 hours*
Monosita tube for	*Esophageal varices*
Warren shunt	*Distal splenorenal shunt*
Bleeding esophageal varices after resuscitation treated with	Sclerotherapy
Primary therapy for prevention of esophageal varices	Sclerotherapy
Compensated liver cirrhosis patient presented with variceal bleed. Treatment of choice	Endoscopic sclerotherapy
Treatment of Choice for Esophageal Varices	Endoscopic Ligation of varices
NOT used as endoscopic sclerosant in treatment of variceal bleeding	Acetic acid
Common operation for bleeding varices	Portocaval shunt
Siguras	*Devascularisation with splenectomy*
NOT a treatment of gastroesophageal varices	Gastric freezing

NODULAR REGENERATIVE HYPERPLASIA

Nodular Regenerative Hyperplasia associated with	Portal Hypertension, Drug induced hepatitis
Nodular regenerating hyperplasia associated with	Drug
NOT true about fibrosis	Nodular regenerative hyperplasia

FOCAL NODULAR HYPERPLASIA

Hepatic lesion diagnosed with high accuracy by using nuclear imaging	Focal nodular hyperplasia
Bull's eye lesion in USG	Focal nodular hyperplasia
Focal nodular hyperplasia	*Typical central scar*
Focal Nodular hyperplasia	NOT more malignant
Hepatic lesion that can be diagnosed with high accuracy by using nuclear imaging	Focal nodular hyperplasia
Liver Lesion diagnosed accurately by Nuclear Imaging	Focal Nodular Hyperplasia
Investigation of choice for focal nodular hyperplasia	*Sulphur colloid*

EXTRAHEPATIC PORTAL VEIN OBSTRUCTION

12 year old boy, hematemesis, malena, mild splenomegaly, no obvious jaundice or ascites	EHPVO

GALL BLADDER

GENERAL FEATURES OF GALL BLADDER

Location of gall bladder	*Junction of right semilunar line with right subcostal margin*
Gall bladder is between	*Right lobe and quadrate lobe*
Vertebral level of Fundus of gall bladder	L1& L2
Sentinel node for gall bladder	Lymph node of lund
Normal capacity of gall bladder	Up to 50 ml
Function of gall bladder	*Acidification of bile*
Bile is concentrated in gall bladder upto	5-10 times
Normal gall bladder epithelium	Simple columnar with brush border
Features of healthy gall bladder on laparotomy	Typical sea green coloured, Wall is thin and elastic
Saint's triad	Gall stones, Hiatus hernia, Diverticulosis coli
Cholesterosis	Diffuse deposition of cholesterol in mucosa of gall bladder
Strawberry gall bladder	Cholesterosis
Cholecystoses refer to	Cholesterosis, Adenomyomatosis, Polyposis
Internal fistula more common between gall bladder and	First part of duodenum
Focal diffuse gall bladder wall thickening with comet tail reverberation artifacts on USG are seen in	Adenomyomatosis of gall bladder
Malignant change in asymptomatic patient with gall bladder polyp	Age > 60, Rapid increase in size of polyp, Size of polyp > 1 cm, Associated gall stones
NOT a feature of malignant transformation of gall bladder polyp on ultrasound	Size of polyp >5 mm
NOT a risk factor for gall bladder carcinoma	Oral contraceptive
Investigation for Visualising Gallbladder	USG
Thickened gall bladder on USG	Acute cholecystitis, Mucosal thickening, Cholesterosis, Ascites

FEATURES OF GALL STONES

Gall stones predisposed by	Choledocholithiasis, Acute cholecystitis, Acute pancreatitis
Predisposing factors of cholelithiasis	*Chronic hemolytic anemia, obesity, pregnancy, high fatty diet*
NOT a risk factor for Cholelithiasis	Pancreatitis
Childhood cholelithiasis	Nieman Pick's disease
Cholelithiasis more common in	Women
Incidence of gallstones high in	Ileal resection
Gall stones	Pigment stones are due to increased excretion of conjugated bilirubin, risk factor for development of gall bladder carcinoma
Treatment of Gall stones	Ursodeoxycholic acid
Gallstones	Increased incidence of stones in diabetics

Gall stones do NOT contain	Oxalates
Percentage of radiopaque gallstones	10%
Radioopacity of gallstones depend on	Calcium
Gallstones	**80% cholesterol, 20% pigment**
Cholesterol stones are	Yellow in colour
Predominant constituent of pale yellow gallstones in gall bladder	Cholesterol
Cholesterol gall stones are due to	Decreased motility of gall bladder, hyposecretion of bile duct, hypercholesterolemia
Brown pigment stones	*Earthy, easily breakable, common in asian population*
Limey bile	Like tooth paste emulsion in gall bladder
Lithogenic bile	Decreased bile and increased cholesterol ratio
Most potent cholerectic	*Bile salt*
Factor in bile juice responsible for preventing the precipitation in cholesterol and formation of gall stones	Bile salts
Substance raised when there is stone in bile duct	Bile salts
Stone formation in gall bladder enhanced by	Clofibrate therapy, Hyperalimentation, Primary biliary cirrhosis, Ileal resection, Cholestyramine therapy
Stone formation in gall bladder is NOT enhanced by	Vagal stimulation
Gall stone formation is NOT influenced by	Hypercholesterolemia
Does NOT lead to pigment stone formation	TPN
MC Site of Gall stone Obstruction	Terminal ileum proximal to Ileocaecal valve
MC Site of Impaction of Gall Stones in CBD	Ampulla of Vater
Mucocele of gall bladder is caused by stone impacted in	Neck of gall bladder
Treatment of choice for mucocele of gall bladder	Cholecystectomy
Management of Gall stone Ileus	Removal of Obstruction
NOT a complication of gallstone	Diverticulosis

DIAGNOSIS OF GALL STONES

Investigation of choice for Gall Stones	USG
Most sensitive method to detect ampullary stone	**Endoscopic ultrasound**
Assessment of Substances in Stones	X-ray Crystallography
Substance used in OCG	Iopanoic acid
Graham Cole test	Oral cholecystography
Contrast used in Graham Cole test	*Iopanoic acid*
OCG	USG has replaced it, Dye used is telepaque
Required for visualisation of gall bladder in oral cholecystogram	Patency of cystic duct, Ability to absorb water, Functioning hepatocytes
CT is least accurate in diagnosis of	Gall stones

TREATMENT OF GALL STONES

Treatment of choice for silent stones in gall bladder	Observation
Incidental gall bladder stone on USG. Asymptomatic	No surgery
Ursodeoxycholic acid	Gall stone dissolving drug
Medical treatment of gall bladder stone is amenable for	Stones less than 15 mm size

Contraindication for medical management of gall stones	Radioopaque stones
50 year old diabetic patient with asymptomatic gallstone(>3 cm) will be best treated by	Early surgery
A young patient, high grade fever with chills, rigor, mild jaundice and acute pain in upper abdomen following cholecystectomy. On examination, she was jaundiced, toxic, hemodynamically stable, vague fullness in upper abdomen. probable diagnosis	Localized collection of bile in peritoneal cavity

FEATURES OF CHOLECYSTITIS

Acute cholecystitis	Boas sign, Murphy sign
Acute cholecystitis is common in	Obese ladies
Acute emphysematous cholecystitis	Clostridium perfringens
Acalculous cholecystitis is caused by	Diabetes mellitus, TPN, Anemia, Tuberculosis, Enteric fever, Leptospirosis, Dengue hemorrhagic fever
Acute acalculous cholecystitis NOT seen in	Malaria
Pain in right hypochondrial region during deep inspiration	Acute Cholecystitis
IgG associated with cholecystitis	**IgG4**
Rokitansky Aschoff sinus is seen in	*Chronic cholecystitis*

DIAGNOSIS OF CHOLECYSTITIS

Recurrent attacks of cholelithiasis, dilated CBD of 1 cm. next line of management	ERCP
Features of cholecystitis on USG	Thick fibrosed gallbladder wall, Stone impacted at neck of gall bladder, Perigallbladder halo
Investigation of choice in acute cholecystitis	USG
Best investigation for acute cholecystitis	HIDA scan

TREATMENT OF CHOLECYSTITIS

Treatment of chronic cholecystitis	Cholecystectomy
After an attack of acute cholecystitis, early suitable period for cholecystectomy	*4 – 6 weeks*
NOT an indication for cholecystectomy	Asymptomatic
Laparascopic cholecystectomy	Erich Muhe
88 year male with end stage renal disease and coronary artery block and metastasis in lung. now presents with acute cholecystitis, patient’s relatives need treatment to do something	Tube cholecystostomy
Contraindications for laparascopic cholecystectomy	Previous laparotomy, Emphysema, Obese individual
Acute cholecystitis.1.5 cm gall stone. symptoms controlled with medical treatment 45 year female	Laparoscopic cholecystectomy immediately
Advantages of laparoscopic cholecystectomy over conventional cholecystectomy	Decrease pain, Decreased stay in hospital, Smaller scar

Open cholecystectomy, 100 ml bile output from drain in first post operative day, abdomen soft. bowel sounds normally heard. best possible advice	Clinical correlation
On 7th postoperative day after laparoscopic cholecystectomy. Patient developed right upper abdominal pain and 10*8 cm collection. treatment consists of	Percutaneous drainage, Laparoscopic cystic duct ligation and percutaneous drain, Roux en Y loop hepatojejunostomy
After cholecystectomy, increased chance of carcinoma	Liver

CARCINOMA GALL BLADDER

Predisposing factors for gall bladder carcinoma	Typhoid cancers, Adenomatous gall bladder polyps, Choledochal cysts, Chronic cholesterol stone, Chronic gall bladder disease, Porcelain gall bladder
Precancerous lesion of gall bladder	Porcelain gall bladder
Organism associated with fish consumption and also causes carcinoma gall bladder	Clonorchis sinensis
Association of carcinoma gallbladder with gall stones	90%
Risk factors for malignant change in an asymptomatic patient with a gall bladder polyp on ultrasound include	Age > 60 years, Rapid increase in size of polyp, Size of polyp > 10 mm, Associated gall stones
NOT a predisposing factor for gall bladder carcinoma	Oral contraceptives
Commonest type of ca gall bladder with gall stones	Adenocarcinoma
Ca gall bladder	Presents with jaundice, Gallstones predispose
Commonest association with carcinoma gall bladder	Secondaries liver
Best investigation modality of gall bladder	USG
Male, laparascopic cholecystectomy, histopathology ca gall bladder T1a	Excision of all port sites
Laparascopic cholecystectomy done in a patient with cholelithiasis. adenocarcinoma with invasion of muscular layer	Wedge hepatic resection with lymph node dissection
Adenocarcinoma of gallbladder infiltrating up to serosa in cholecystectomy specimen	Radical cholecystectomy
Survival in unresectable ca gall bladder	4-6 months

BILIARY TRACT

ANATOMY OF BILIARY TRACT

Common bile duct	Opens 10 cm distal to pylorus, Lies anterior to IVC, portal vein lies posterior to it, Open to duodenum after JOINING with main pancreatic duct, Anteriorly related to first part of duodenum, Related to IVC posteriorly
Common bile duct	Lies in free margin of lesser omentum, Right to hepatic artery, Anterior to portal vein, Open in second part of duodenum
Common bile duct is related to	Portal Vein

Common bile duct is related to	Head of pancreas
Relation of common bile duct	Lies right to hepatic artery
Sphincter of Oddi contains	3 sphincters
Spiral Valve of Heister is seen in	Cystic Duct
Spiral valve in	*Neck of gall bladder*
Predominant blood supply to supraduodenal bile duct is derived from	Vessels than run upward along the bile duct from duodenal end of duct such as retroduodenal and gastroduodenal arteries

FEATURES OF BILIARY TRACT

Size of CBD	7.5 cm long
Naloxone reduces biliary colic caused by	Morphine, fentanyl, pethidine
Biliary obstruction caused by	Clonorchis, ascaris, fasciola
Low serum haptoglobin in hemolysis is masked by	Bile duct obstruction
Bile salt induced is reduced by administration of	Cholestyramine
Central dot sign	Caroli's disease
Double duct sign	Combined biliary and pancreatic duct obstruction
Vanishing bile duct syndrome	Sarcoidosis
Bile ductopenia is seen in	GVHD
Yellow discoloration of umbilical region in patients with ruptured common bile duct is known as	*Ransohoff's sign*
MC bile duct tumour	Adenocarcinoma
MRCP	MRI is used to obtain image, It shows biliary tree
Best suture for common bile duct	Non synthetic absorbable

CHOLANGITIS

MC cause of cholangitis	CBD stone
MC cause of suppurative cholangitis	Stone in common bile duct
MC Organism cultured from Bile in Cholangitis	E.coli, 2nd Klebsiella
Acute cholangitis following ERCP is caused by	E.coli
MC cause of Obstruction in Cholangitis	Gall stones
In case of choledochal cyst, bile diversion to small intestine NOT done because of risk of	Recurrent cholangitis
Organism associated with acute cholangitis	E.coli
Cholangitis	Increased leucocyte count, Increased transaminases, Increased bilirubin, Increased alkaline phosphatase, Association with chills and fever
Primary sclerosing cholangitis	Cirrhosis occur, Fibrosing cholangitis of bile duct, Periductal onion skin appearance, Jaundice, Associated with ulcerative colitis
Primary Sclerosing Cholangitis	Inflammation of Extrahepatic bile duct
Primary sclerosing cholangitis	More common in males, Both intra and extra hepatic ducts, ERCP is a sensitive investigation
A pt presents with history of diarrhea for several years with recent onset pruritis and increased alkaline	Sclerosing cholangitis

phosphatase, normal SGOT/PT and USG shows no gall stones and biliary tract abnormality, diagnosis	
NOT true about primary sclerosing cholangitis associated with ulcerative colitis	PSC reverts after total colectomy
Unrelated to clinical or histological picture	*Presence of anti mitochondrial antibody in high titre*
Air in biliary tract NOT seen in	Sclerosing cholangitis
Charcot triad	Pain, fever and jaundice
Reynold's pentad	Cholangitis
Reynold's pentad of toxic cholangitis is associated with	Confusion, Septic shock
Smoking protective in	Primary sclerosing cholangitis
Skip Lesions	Primary sclerosing Cholangitis
Increased copper excretion in urine NOT seen in	Primary sclerosing cholangitis
IOC in acute cholangitis	HIDA scan
ERCP is NOT used for diagnosis of	Cholangitis
Ultimate treatment of primary sclerosing cholangitis	Liver transplantation

FEATURES OF COMMON BILE DUCT STONES

MC type of CBD stone	Secondary (from Gall bladder)
Stones in common bile duct	Present with charcot triad, suggested by bile duct diameter > 6 mm of ultrasound, ERCP, Sphincterotomy and balloon clearance is the standard treatment
CBD stone present with	Increased bilirubin, Increased WBC, Increased liver enzymes, Fever with rigors
Meniscus sign	Impacted Stone in CBD
Seagull/Mercedes Benz/Crow foot sign	Radioluscent Gall Stone with Gas
Aminopeptidase elevated in obstruction of	CBD
An ultrasound examination shows dilated intrahepatic biliary channels with a small gall bladder. Most likely possibility	Common bile duct stone
NOT true about CBD stone	Origin always in gall bladder
NOT a manifestation of CBD stone	Distended gall bladder

DIAGNOSIS OF COMMON BILE DUCT STONES

First Investigation for CBD Stone	USG
Obstruction in CBD is best diagnosed by	ERCP
Most appropriate for diagnosis of CBD stone	ERCP
Investigation for CBD Stone	ERCP
If ERCP is failed in evaluation of bile duct	**Percutaneous transhepatic cholangiogram**
Non invasive investigation for CBD Stone	MRCP
In cholangiography, CBD stone appear as	Meniscus sign
Dye useful in IV cholangiography	Biligraffin

TREATMENT OF COMMON BILE DUCT STONES

Burhenne procedure	*Extraction of CBD stone through T-tube*
Stones in bile duct	ERCP and bile duct stone extraction
A patient having multiple gall stones and show 8 mm dilatation and 4 stones in CBD, best treatment modalities	Cholecystectomy and choledocholithotomy at same setting, Sphincterotomy and then cholecystectomy
Treatment of choice for symptomatic retained CBD stone	Endoscopic sphincterotomy
Ideal treatment of stenosis in sphincter of Oddi	Endoscopic sphincterotomy
Treatment of 8 mm retained CBD stone	Endoscopic sphincterotomy
A retained stone impacted in distal common bile duct seen on T tube cholangiogram. best management of stone	Endoscopic sphincterotomy and stone extraction
One year after cholecystectomy patient presented with 2.5 cm size stone in CBD, treatment of choice is	Endoscopic sphincterotomy with stone extraction
Treatment of CBD stone	ESWL, Exploration of bile duct and recovery of stone, Bile duct stenting, Nasobiliary drainage, Percutaneous drainage
5 days after CBD surgery, there is small leak. best treatment	ERCP and stenting
Absolute indication for choledochotomy	Palpable CBD stone
Procedure of choice for elective removal of CBD stones for most of patient	Endoscopic choledocholithotomy
NOT an absolute indication of choledochotomy	Multiple stones in gall bladder
After exploration of common bile duct, T tube is removed on	12th postoperative day
Cholangiography via T tube done after ____ days of cholecystectomy	10-14 days
T tube removed on	4-6 weeks
Postoperative cholangiogram via T tube is done following Choledochotomy	14 days

BILE DUCT INJURIES

Bile duct injury	Incidence following open cholecystectomy is in range of 0.2-0.3%, Incidence following laparoscopic cholecystectomy is three times higher than the open cholecystectomy, Untreated cases may develop secondary biliary cirrhosis
Bile duct injury commonly associated with	Laparoscopic cholecystectomy
In bismuth/Strasberg classification, cystic blow out is classified as	Type A
Lateral injury to common bile duct in Bismuth/Strasberg classification	*Type D*

BILIARY ATRESIA

Extrahepatic biliary atresia	Alcoholic stool, Conjugated hyperbilirubinemia, Absence of nucleoid in duodenum in HIDA scan, Jaundice is presenting feature
Triangular cord sign	*Extrahepatic biliary atresia*
In neonatal cholestasis, if serum gamma glutamyl transpeptidase is more than 600 IU/L. most likely diagnosis	Biliary atresia
No risk of kernicterus in	*Biliary atresia*
Histological features of extrahepatic biliary atresia	Marked bile duct proliferation, Fibrosis of hepatic duct, Parenchymal cholestasis
Better prognostic factor for operation of biliary duct obstruction in newborn	Size of ductile > 200 micron, Age > 8 weeks
2 month baby, history of jaundice. Turmeric coloured urine, pale coloured stools. examination reveals liver span of 10 cm. firm in consistency, spleen 3 cm. most specific investigation	Peroperative cholangiogram
Gold standard for definitive diagnosis of extrahepatic biliary atresia	Preoperative cholangiography
HIDA scan most useful in diagnosis of	Biliary atresia

CHOLEDOCHAL CYST

Choledochal cyst	Excision is ideal treatment, Epigastric mass, Jaundice, Pain in abdomen
Todani classification for	*Choledochal cyst*
MC type of choledochal cyst	Type I
Saccular(fusiform) diverticulum of extrahepatic bile duct in choledochal cyst is classified as	Type II
Choledochocele	*Type III choledochal cyst*
Type V choledochal cyst	*Caroli's disease*
Caroli disease	*Congenital hepatic fibrosis is associated with it, central dot sign, diagnosed by ERCP or PTC*
Clinical feature of choledochal cyst in adult	Pain, lump and progressive jaundice
NOT true about choledochal cyst	Type II is most common
Treatment of choice for choledochal cyst	Excision
In case of choledochal cyst, bile diversion into small intestine is NOT done because of risk of	Recurrent cholangitis

BILIARY STRICTURE

Causes of bile duct strictures	Bile duct carcinoma ,Chronic pancreatitis, Trauma, CBD stone
MC cause of bile duct stricture	Post cholecystectomy
Biliary strictures developing after laparascopic	Upper part of common bile duct

cholecystectomy in	
Initial investigation of post cholecystectomy biliary stricture	Magnetic resonance cholangiography

BILIARY FISTULA

MC cause of biliary fistula	Post surgical
Does NOT contribute to enterobiliary fistula	Gastric ulcer

CHOLANGIOCARCINOMA

Increased incidence of cholangiocarcinoma is seen in	Polycystic disease of liver, sclerosing cholangitis, liver flukes
Predisposing factors for cholangiocarcinoma	Ulcerative colitis, Clonorchis sinensis, Choledochal cyst, Primary sclerosing cholangitis, Polycystic disease of liver, Chronic typhoid carrier
NOT a major risk factor for cholangiocarcinoma	Choledocholithiasis
NOT a predisposing factor for cholangiocarcinoma	Chronic pancreatitis, CBD stone, Hydatid cyst of liver
Does NOT predispose to cholangiocarcinoma	Chronic pancreatitis
MC site of cholangiocarcinoma	Hilum
Most common type of cholangiocarcinoma	*Type II (junction of CBD and hilum)*
Type II cholangiocarcinoma	*Involve bifurcation without involvement of secondary intrahepatic ducts*
Klatskin tumour	Junction of bile duct and hilum of liver
Site of klatskin tumor	Confluence of right and left hepatic duct
Criteria for unresectability in cholangiocarcinoma	*Bilateral hepatic duct involvement upto secondary radicals, bilateral hepatic artery involvement, distant metastasis, encasement of portal vein distal to its bifurcation*

PNEUMOBILIA

Air in biliary tract	Gall stone ileus, Carcinoma gall bladder, Endoscopic papillotomy
Air in biliary tract NOT seen in	Sclerosing cholangitis
Pneumobilia	Sphincterectomy, Rupture of hydatid cyst, Gall stone ileus
NOT associated with pneumobilia	Mirizzi syndrome

SPLEEN

ANATOMY OF SPLEEN

Spleen originates from which space of peritoneal cavity	Dorsal mesentery
Spleen projects into which space	Greater sac
Red pulp of spleen	Vascular sinuses
White pulp of spleen	Periarteriolar lymphoid sheath, B cells, Antigen presenting cells
NOT a component of white pulp of spleen	Vascular sinus
Other than spleen, occlusion of splenic artery at its origin will most likely affect the blood supply of	*Fundus of stomach*
Splenic artery DOES NOT supply	Liver
Spleen derives nerve supply from	Celiac plexus
Accessory spleen found at	Hilum, Tail of pancreas, Greater omentum,small bowel mesentry
Commonest sign of accessory spleen(spleniculi)	Hilum of spleen
Accessory spleen NOT found at	Presacral area

FEATURES OF SPLEEN

Ivermark syndrome	*Asplenia syndrome*
Removal of senescent RBC from circulation by spleen	Culling
Spleneculli	Hypersplenism
Hypersplenism associated with	Pancytopenia
Splenosis means	Rupture of spleen and distribution of its tissue on peritoneum
Right isomerism	Asplenia
Percentage of total blood volume in spleen	2%
MC cyst of spleen	Hydatid cyst
MC tumor of spleen	Lymphoma
MC primary lymphoma of spleen	Small lymphocytic lymphoma
Splenic vein thrombosis best treated by	Splenectomy
MC cystic disease of spleen	*Pseudocyst*
Splenomegaly in splenic abscess	*Rare*
Wandering spleen	*Congenital atresia of dorsal mesogastrium in children, spleen is attached to long vascular pedicle without usual mesenteric attachment, torsion and infarction are common, splenectomy is not always required*
Polysplenia syndrome	*Absence of a part of inferior vena cava, bilateral left lung morphology, associated with congenital heart diseases*

SPLENOMEGALY

Tropical splenomegaly is seen in	Malaria
Tropical splenomegaly	*Parasites absent in peripheral blood*
NOT a feature of Tropical Splenomegaly	Persistent high grade fever
Does NOT cause increase in size of spleen in later stages	Sickle cell anemia
Treatment of tropical splenomegaly	*Chloroquine, proguanil*
Splenomegaly is common in sub acute endocarditis of	*Longer duration*

SPLENECTOMY

MC indication of splenectomy in India	Trauma
Splenectomy is most useful in	Hereditary spherocytosis
Splenectomy is indicated in	Primary hypersplenism, Hereditary spherocytosis, ITP, Portal hypertension, Aplastic anemia, Lymphoma, CML
Indication for splenectomy in ITP	*ITP has been diagnosed for 6 weeks, still platelet count is less than 10,000 with or without bleeding, ITP has been diagnosed for 3 months with platelet count less than 30,000*
Splenectomy is least useful in	Thalassemia major
Absolute indication for splenectomy	Splenic abscess, Hereditary spherocytosis, Fibrosarcoma
NOT an indication of Splenectomy	Splenorenal shunt
NOT an absolute indication for splenectomy	Autoimmune hemolytic anemia
MC incision for splenectomy	*Midline incision*
Best time for platelet infusion in an ITP patient planned for splenectomy	After ligating splenic artery
An evidence that splenectomy might benefit a patient with idiopathic thrombocytopenic purpura includes	Increase in platelet count of corticosteroid therapy
MC infection after splenectomy	Pneumococcal(Sterptococcus pneumonia)
MC infection after Splenectomy	Capsulated bacteria
MC complication after splenectomy	Left lower lobe atelectasis, Chest infection
MC complication of splenectomy	Pulmonary complications
OPSI	Begins with mild appearing prodrome, May present with septic shock, Usually does not respond to antibiotic treatment, Develops 1-5 years after splenectomy
NOT true about OPSI	Maximum risk is within 1 years of splenectomy
NOT a post splenectomy infection	Herpes zoster
Pneumococcal vaccine most effective	When given preoperatively

SPLENIC INJURIES

Kehr sign	Splenic injury
Splenic injury, conservative management done in	Young patient

Grade III splenic laceration, hemodynamically stable	Conservative management
Splenic injury diagnosed on X ray by	Obliteration of splenic shadow, Rib fracture

SPLENIC RUPTURE

Grade IV splenic injuries	*Hilum and vessels*
Grade V splenic injuries	*Shattered spleen*
Kehr sign in splenic rupture is	Pain over left shoulder
Features of Splenic rupture	Elevation of Left diaphragm, Obliteration of Psoas shadow & Splenic Outline
Most important radiological sign of splenic rupture	Obliteration of splenic shadow
NOT a feature of splenic rupture	Obliteration of colonic air bubble
Investigation of choice for diagnosis of splenic rupture	CT scan
Splenic rupture in children is best diagnosed by	Tc 99 scan
Accidental splenic rupture is treated with	Catgut suturing with omental patch

PANCREAS

ANATOMY OF PANCREAS

Posterior surface of neck of pancreas is related to	Superior mesenteric vein
Tail of Pancreas has	More Islets of Langerhans
Posterior relation of pancreas	Common bile duct, Aorta, Inferior vena cava
NOT a posterior relation of pancreas	First part of duodenum
Branching pattern of duct of pancreas	*Herring bone pattern*
Duct NOT found in Pancreas	Bellini duct
Pancreas receives its blood supply from	*Celiac and superior mesenteric artery*
Anterosuperior and posterosuperior pancreaticoduodenal arteries are derived from	*Celiac artery*
Anteroinferior and posteroinferior pancreaticoduodenal arteries are derived from	*Superior mesenteric artery*
Blood and tail of pancreas is supplied by	*Splenic artery*

FEATURES OF PANCREAS

MC embryological abnormality of pancreas	*Divisum*
Treatment of congenital cyst of head of pancreas	Total excision
Amylin is secreted by	Beta cells of pancreas
True increase in islet cell	Nesidioblastoma, Insulinoma
NOT a feature of pancreatic ascites	Low protein

Lymphocytic infiltration in islet cells of pancreas characteristically seen in	Juvenile diabetes
Somatostatinoma and non functional PET are common in	*Head*
Management of pancreatic abscess	External drainage
Child presents with increased fecal fat excretion and increased fetal nitrogen levels	Pancreatic insufficiency
MC site of ectopic pancreatic tissue	Stomach
Islet tissue in ectopic pancreas is present only in	*Stomach*
Autoimmune pancreatitis	*Presents with obstructive jaundice, increased IgG4*
Mucinous cyst of pancreas	*Premalignant lesion, columnar epithelium lesion, microcystic adenoma*
Increased amylase seen in	Pancreatic pseudocyst, Perforated peptic ulcer, Ruptured ectopic pregnancy
Serum amylase raised in	Blocked salivary duct, Ruptured ectopic, Pancreatitis
Serum amylase NOT increased in	Appendicitis
Bentiromide test for	**Pancreatic function**
Isotope used for pancreatic scanning	Selenium 75 methionine
Most worthwile investigation for detection of suspected pancreaticobiliary worms	*ERCP*
Somatostatinoma is associated with	*Hypochlorhydria*
Somatostatinoma causes	*Watery diarrhea*
Investigation of choice for both primary and secondary neuroendocrinal tumors	*Somatostatin receptor scintigraphy*
Radionucleides used in treatment of neuroendocrinal tumors	*Iridium DTPA, Ytrrium 90, 177 Lu coupled analogues*
NOT a treatment of somatostatinoma	*Radiotherapy*

FEATURES OF ACUTE PANCREATITIS

MC cause of Acute Pancreatitis in Children	Trauma
MC cause of acute pancreatitis	Gall stone
MC cause of acute pancreatitis	Biliary tract disease
Pancreatitis caused by	Gallstones, Alcohol, Hypercalcemia, Hypertriglyceridemia
Drugs causing pancreatitis	Azathioprine, L asparaginase, Furosemide
Pancreatitis occur with	Furosemide, valproic acid, didanosine
Acute and recurrent pancreatitis is reported to occur in	Isovaleric aciduria
NOT a cause of acute pancreatitis	Hemochromatosis
Not a cause of Pancreatitis	Islet cell hyperplasia
Obesity is NOT associated with	Pancreatitis
MC Complication of ERCP	Acute Pancreatitis
Type of Pancreatitis having best prognosis	Gall stone pancreatitis
Signs of Acute Pancreatitis	Grey Turner – Discolouration In Flanks, Cullen – Discolouration Around Umbilicus
Features of acute pancreatitis	*Sentinel loop, colon cut off sign, renal halo*

	sign
Discolouration of Flanks in Acute Pancreatitis	Grey Turner Sign
Acute pancreatitis	Pain lasting for several hours
Hypocalcemia in Acute pancreatitis	Severe form of disease
MC complication of acute pancreatitis	Pseudocyst of pancreas
Cause of acute loss of vision in a patient of alcoholic pancreatitis	Purtscher retinopathy
MC cause of death in early stage of acute pancreatitis	*Respiratory complications*
MC cause of death in patients with acute pancreatitis	*Secondary infections*
Common cause of death in acute pancreatitis	Shock
NOT true about acute pancreatitis	Low serum lipase
NOT a feature of acute pancreatitis	Hypercalcemia
NOT seen in acute pancreatitis	Hypoglycemia
Vascular complications of acute pancreatitis	Splenic vein thrombosis, Splenic artery thrombosis, Gastroduodenal artery aneurysm
Poor prognostic factor in acute pancreatitis	Decreased serum calcium, Increased blood sugar
Poor prognosis	Decreased calcium, Decreased PaO2, Hypocalcemia, Hyperglycemia
Poor prognostic factor in a patient with Pancreatitis includes	Leucocytosis > 20,000 / microlitre
Ranson's criteria	At the time of admission, During initial 48 hours
Glasgow prognosis (Imrie scoring)	*Acute pancreatitis*
NOT a poor prognostic factor	Hyperamylasemia
NOT a bad prognostic feature in acute pancreatitis	Serum amylase > 1000 IU/ml
Acute Pancreatitis	S. Lipase
Severity of pancreatitis does NOT correlate with	Amylase
NOT used to predict severe acute pancreatitis	CRP <100
Severity of pancreatitis is NOT associated with	CPK
NO SIGNIFICANT CORRELATION between Acute Pancreatitis and	Serum Amylase
NOT a Ranson's criteria	Serum amylase
NOT included in Ranson's criteria	Prothrombin time

DIAGNOSIS OF ACUTE PANCREATITIS

Colon Cut off Sign, Gasless Abdomen	Acute Pancreatitis
Raised in acute pancreatitis	*IL-10*
Investigation of choice for Acute Pancreatitis	CT Scan
Pancreatitis after 5 days useful investigation	LDH
Patient presents with pain radiating to back. investigation of choice	CT scan
Acute pancreatitis	CECT preferred over USG for diagnosis
NOT a CT scan feature of acute pancreatitis	Dilated main pancreatic duct
ERCP in pancreatitis is to rule out	Gall stones, Pancreatic divisum, Annular pancreas

TREATMENT OF ACUTE PANCREATITIS

Medical treatment of pancreatitis	Calcium
Used in management of acute pancreatitis	*Gabexate, somatostatin, peritoneal dialysis*

FEATURES OF CHRONIC PANCREATITIS

Chain of Lake appearance (ERCP)	Chronic pancreatitis
NOT a feature of chronic pancreatitis	Paralytic ileus
NOT a feature of chronic calcific pancreatitis	Hypercalcemia
TIGAR – O classification for	**Chronic pancreatitis**
Chronic alcoholic abdominal pain radiating to back responds to analgesics. on evaluation pancreatic duct found to be dilated and stones in tail of pancreas	Medical management

MANAGEMENT OF CHRONIC PANCREATITIS

Chronic alcoholic, abdominal pain responding to analgesic. pancreatic duct found to be dilated stones noted in tail of pancreas	Medical treatment
Pain Relief in Chronic Pancreatitis can be obtained by destruction of	Celiac Ganglia
Chronic alcoholic, intractable abdominal pain. pancreatic duct found to be dilated stones noted in tail of pancreas	Pancreaticojejunostomy
50 year female, 2 year history of recurrent abdominal pain with radiation to back, pain refractory to simple analgesics, CECT dilated pancreatic duct	Longitudinal pancreatic jejunostomy
Procedures for chronic pancreatitis	*Puestuous, headcoring, Berger procedure, Frey Frey procedure*
Prolonged and complicated pancreatic surgery done for chronic pancreatitis. most preferred route of nutrition	Feeding jejunostomy

PSEUDOCYST OF PANCREAS

Pseudocyst of pancreas commonly occur after	Pancreatitis
MC cause of Pseudocyst Pancreas	Pancreatitis, 2nd Trauma
MC cause of Pseudocyst of Pancreas in Children	Trauma
MC site of pseudocyst of pancreas	Body and tail of pancreas
Pseudocyst of pancreas is located on	*Lesser sac*
Nealon classification is for	*Pseudocyst of pancreas*
Pseudocyst of pancreas	It can present from mediastinum to pubis, Contains a wall of fibrous granulation tissue
A 20 years old football player received hard kick in	Pseudopancreatic cyst

epigastrium. A large cystic swelling appeared in epigastrium 2 weeks later. Most likely diagnosis	
Swelling in pancreatic pseudocyst	Below umbilicus
NOT true about psedopancreatic cyst	MC site is head of pancreas
NOT true about pseudopancreatic cyst of pancreas	Drains into duodenum
NOT true about pseudocyst of pancreas	Percutaneous aspiration is the treatment of choice
MC Complication of Pancreatic Pseudocyst	Infection
NOT a serious complication of pseudocyst of pancreas	Calcification of cyst wall
Least complication of Pancreatic Pseudocyst	Carcinomatous change
After 3 weeks of duration pancreatic pseudocyst 5 cm in size should be managed by	USG an followup
Indications of surgery in pseudocyst of pancreas	Persistent, Infective
Treatment of pseudopancreatic cyst	Endoscopic drainage, Percutaneous drainage, Internal drainage, Pancreatectomy
Treatment of Choice for Uncomplicated Pancreatic Pseudocyst	Internal Drainage (Cystojejunostomy,Cystogastrostomy, Cystoduodenostomy)
Walled off necrosis is similar to	**Pancreatic pseudocyst**
NOT a treatment of pseudocyst of pancreas	*Removal of involved segment of pancreas*

HEMORRHAGIC PANCREATITIS

L-Asparaginase, Tapsoglutide (GLP 1 analogue)	Hemorrhagic Pancreatitis
Cullen sign	Acute hemorrhagic pancreatitis

FEATURES OF CARCINOMA PANCREAS

MC genetic aberration in pancreatic cancer	**K ras**
Risk factor for Pancreatic carcinoma	Smoking
Pancreatic carcinoma is caused by	Clonorchis
Pancreatic carcinoma	P53 mutation associated with 75% cases, hereditary pancreatitis significantly increase risk, Obstruction of bile and pancreatic secretion is common, Smoking predispose
MC Pancreatic Cancer	Ductal Adenocarcinoma
MC site of Ca Pancreas	Head
Carcinoma pancreas attains greatest size when it is located in	Body and tail
Pancreatic Carcinoma is associated with	SIADH
Migratory thrombophlebitis	Carcinoma pancreas
MC Migratory Thrombophlebitis associated with	Pancreatic Carcinoma
Serous cystadenoma of pancreas	*Central calcification, stellate scar*
NOT true about mucinous cystadenoma pancreas	Microcystic adenoma
Inverted 3 sign	Ca head of pancreas
Scrambled egg appearance	Pancreatic carcinoma
Widening of C loop of duodenum is a feature of	Pancreatic head overgrowth
Duodenal C loop with irregular mucosal pattern on upper gastrointestinal barium series	Carcinoma head of pancreas

Spongy appearance with central sunburst calcification	Serous cysadenoma of pancreas
Multicystic Mass Bunch of Grape Appearance, Dilated Pancreatic Duct	Intraductal Papillary Mucinous Neoplasm
Fish eye lesion	*Intraductal papillary mucinous neoplasm*

MANAGEMENT OF CARCINOMA PANCREAS

Most sensitive investigation of Carcinoma Pancreas	ERCP
Investigation to diagnose carcinoma head of pancreas	Hypotonic duodenogram, USG, Endoscopy, CT scan
Drug used for Ca Pancreas	5-Fluorouracil/Gemcitabine
Chemotherapeutic agent of choice in ca pancreas	5-fluorouracil
Gemcitabine is effective in	Pancreatic cancer
Side effect of gemcitabine	**Flu like syndrome**
Preferred procedure for non resectable carcinoma head of pancreas	Cholecystojejunostomy
Longitudinal pancreaticojejunostomy with preservation of head of pancreas	Whipple procedure
NOT resected in whipple operation	Portal vein
MC surgical complication of Whipple procedure	Disruption of gastric anastomosis
Advantage of bladder drainage over enteric drainage after pancreatic transplantation is better monitoring of	Amylase levels
Prevention of pancreatic carcinoma	Urgent weight reduction, Strict vegetarian diet, Stop cigarette smoking

PERIAMPULLARY CARCINOMA

Site having best prognosis in ca Pancreas	Periampullary
Reverse three sign of frostberg	Periampullary carcinoma
Obstructive jaundice, weight loss. CT scan CBD dilated till lower end main pancreatic duct is also dilated. Pancreas normal Obstructive jaundice, weight loss. CT scan CBD dilated till lower end main pancreatic duct is also dilated. pancreas normal	Periampullary carcinoma
Periampullary carcinoma feature is	Early jaundice
Double duct sign	*Periampullary carcinoma*
Silvery, shiny clay coloured stool is associated with	*Periampullary carcinoma*

PANCREATIC INJURIES

Pancreatic injury	Majority of post op complications are due to missed duct injury, Commonly associated with vascular injury, Amylase increased in 90% of patient, HRCT is investigation of choice
NOT true about pancreatic injury	Fracture is common at junction of head and body, Peritoneal lavage is good for making diagnosis

FEATURES OF GASTRINOMA

MC endocrine tumour in MEN I	Gastrinoma
Zollinger Ellison syndrome is due to tumour of	G cells
Zollinger Ellison Syndrome arise from	Non beta cells of pancreas
MC site of gastrinoma	*Duodenum*
MC Site of Zollinger Ellison Syndrome	Duodenal Loop
70-90% gastrinoma occur in	Passaro triangle
Triad of Zollinger Ellison syndrome	Peptic ulceration, gastric hypersecretion,Non beta cell tumour
MC symptom of carcinoid	**Abdominal pain**
Zollinger ellison syndrome	Diarrhoea, Post bulbar ulcer, Recurrent duodenal ulcer
Zollinger Ellison syndrome	Gastrin levels > 1000 pg/ml, BAO > 15 meq/hr, Somatostatin inhibits hcl, Omeprazole helpful, Secretin increases gastrin secretion in Zollinger Ellison syndrome, MC site duodenal loop, It is an endocrine disease, 50% malignant, Surgical excision is done
MC symptom of gastrinoma	*Abdominal pain*
Feature of gastrinoma	*Secretes multiple hormones*
MC manifestation of Zollinger Ellison Syndrome	Diarrhoea
Intractable peptic ulcer	Zollinger Ellison Syndrome
Multiple gastric ulcers, hypertrophied mucosa	Zollinger Ellison syndrome
MC hormone Secreted by Zollinger Ellison Syndrome	Gastrin, 2nd ACTH
Diarrhea in ZES due to	Gastrin
Gastrinoma is associated with	*Increased parietal cells*
NOT true about Zollinger Ellison Syndrome	Pyloric antral tumour
NOT true about zollinger Ellison syndrome	Tumor of beta cells of pancreas
NOT true about ZES	Exocrine
Zollinger Ellison syndrome is NOT characterized by	Massive HCL in response to histamine injection
NOT a feature of Zollinger Ellison syndrome	Beta cell tumours of pancreas
Normal basal acid output	5 – 10 m mol/hr
Basal acid output is increased by	*Alkalinization of antrum*
Increased ratio of basal acid output to maximal acid output	Zollinger Ellison Syndrome
12 hours nocturnal basal output in ZES	400 ml
Normal fasting gastrin levels	*50 mg/L*
Fasting gastrin levels	<120 pg/L
Fasting gastrin level in gastrinoma	**>150 – 200 pg/ml**
Most sensitive imaging study for primary gastrinoma	**Endoscopic USG**
Imaging investigation for secondary gastrinoma	**Octreoscan**
NOT true about diagnosis of ZES	Serum gastrin value of 200 pg/ml with secretin stimulation
Does NOT occur in patient with gastrinoma	Basal acid output less than 15 mEq/L

MANAGEMENT OF GASTRINOMA

Most Sensitive & Specific for Zollinger Ellison Syndrome	Secretin test
Secretin test	*Act by direct stimulation of pancreas,*

	may be a dissociation between the results of secretin test and other tests of absorptive function, secretin test measures the secretory capacity of ductular epithelium
Most important test for diagnosis of Zollinger Ellison syndrome	Secretin injection test
Diagnosis of Zollinger Ellison syndrome	Basal acid output 15 meq/hour, serum gastrin 500 pg/ml. ulcers in proximal jejunum and lower end of esophagus
Treatment of choice in Zollinger Ellison syndrome	PPI
Protein pump inhibitor	Omeprazole
Drug of choice for ZES	Omeprazole
Omeprazole	Negative gastric acid pump,block H+ K+ ATPase pump
NOT true about omeprazole	*Requires dose reduction in renal failure*
Treatment of Zollinger Ellison syndrome	PPI, H2 blockers, Somatostatin
Treatment of ZES	Excision of tumor alone

FEATURES OF INSULINOMA

MC Pancreatic Endocrine Neoplasm	Insulinoma
Common endocrine tumour of pancreas arise from	Beta cells
MC endocrine tumor of pancreas	Insulinoma
MC islet cell tumour	Insulinoma
MC Site of Insulinoma	Equally Distributed
Insulinomas are usually	Single
Insulinoma	Insulin level = C peptide level
Weakness, tachycardia, palpitation, gidiness, sweating relieved on taking food	Insulinoma
Signs of hypoglycemia in insulinoma, if glucose level falls below	*2.8 mmol/L*
Weight loss NOT seen in	Insulinoma
Insulinoma PARADOXICALLY cause	Weight gain
Whipple triad	Islet cell tumor
Whipple's triad of Insulinoma	Fasting hypoglycemia, symptoms of hypoglycemia, immediate relief of iv glucose

MANAGEMENT OF INSULINOMA

Factitious hyperinsulinemia is differentiated from Insulinoma by	C-peptide
Important investigation in insulinoma	Endoscopic ultrasound
Most sensitive investigation for Insulinoma	72 hours Fasting Test
Most sensitive investigation for insulinoma	*Intraoperative ultrasound*
Ocreotide used in	Insulinoma, glucagonoma, carcinoids
Diazoxide	K+ channel opener, can be used as hypertensive agent,

	used in insulinoma
Treatment of insulinoma	Enucleation
Treatment of insulinoma	**Diazoxide, Everolimus (mTOR inhibitor)**
NOT a treatment for Insulinoma	Pancreatectomy

GLUCAGONOMA

Glucagonama	Necrotising migratory erythema (not pathognomic), annular erythema
Migratory necrolytic erythema	Glucoganoma syndrome
Glucagonoma is associated with	*Weight loss*
Glucagonoma is common in	*Tail*
Glucagonoma are actually	*Large tumor (5 – 10 cm)*

MUCOVISCOIDOSIS

In mucoviscoidosis of pancreas, commonest defect in	Ileum
Mucoviscoidosis affects the epithelium of	Respiratory tract, Reproductive tract, Gastrointestinal tract
Mucoviscoidosis commonly related to	Fibrocystic disease of pancreas

ANNULAR PANCREAS

Annular pancreas	Complete or partial rotation failure, Congenital, Normal histology, Surgical correction should be done
Treatment of annular pancreas	Duodenojejunostomy

PANCREATIC CHOLERA

Pancreatic cholera	Hypokalemia, Hypochlorhydria, Hypercalcemia, Hypercalcemia, Glucose intolerance
NOT true about pancreatic cholera	Hypocalcemia

DRUGS ACTING ON GASTROINTESTINAL TRACT

Aprepitant	NK 1 antagonist, Crosses blood brain barrier, Ameliorate nausea and vomiting induced by CT, Metabolized by CYP450
Receptor for vomiting	5HT3
Antiemetic used in vomiting induced by anticancer drugs	Ondansetron
For chemotherapy induced vomiting, 5HT3 antagonist having maximum potency	Ondanseton
Ondansetron acts by	5HT3 antagonism, Acting on CTZ

Granisetron	5HT3 antagonist
Antiemetic action produced through	Decreased CTZ stimulation, 5HT4 agonistic action
Drug NOT acting as prokinetic by stimulating 5HT4	Domperidone
Metoclopramide	Enhances colonic motility
NOT an antiemetic	Cinnarizine
Drug prolonging QT interval	Cisapride
Cisapride is contraindicated in	GI obstruction, hemorrhage, pregnancy
Bisacodyl	Stimulant purgative
Cabenoxolone sodium	Promotes ulcer healing, Slows down prostaglandin degradation, Derived from glycyrrhetinic acid
NOT the effect of ranitidine as compared to cimetidine	Anti androgenic action
Cimetidine does NOT inhibit metabolism of	Ketoconazole
NOT used for motion sickness	Metoclopramide